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KERNICTERUS

A Follow-Up Study of Thirty-Five Erythroblastotic Infants

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WITH the advent of more detailed knowledge concerning the pathogenesis and treatment of erythroblastosis fetalis, interest has been focused on the possible untoward effects of a successful therapeutic regimen that may result in a live but helpless infant with kernicterus.

The term "kernicterus" was devised by Schmorl [1] in 1903 in referring to what Orth [1] had called "nuclear jaundice" in 1875. Orth described a primary necrosis of the brain associated with secondary bile staining and degeneration of the ganglion cells. In 1907 Bencke summarized the prevailing views concerning the relationship between cerebral injury and kernicterus, and it is of interest that one of these theories, that damage to ganglion cells is caused by ischemia (or trauma), in which area of damage there is a subsequent pigmentation, has gained wide acceptance among present day workers in the field. In 1932 Diamond and his colleagues [2] linked together as a single entity the heretofore separate disease states of icterus gravis, congenital fetal hydrops and congenital anemia of the newborn. The new entity was called "erythroblastosis fetalis." In 1933 Zimmerman and Yannet [3] commented on the association between those children jaundiced by the second day of life who died by the fifth or sixth day with signs of involvement of the central nervous system and others who, after a similarly severe attack of neonatal jaundice, lived, only to show later mental retardation and symptoms associated with injuries to basal ganglions (i. e. spasticity of an extrapyramidal type, athetoid and choreiform movements and emotional instability). They noted that in those cases that showed kernicterus at autopsy there was discernible on microscopic section bile pigment in isolated nerve cells in the cerebral

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1. Cited by Zimmerman and Yannet.[3]

2. Diamond, L. K.; Blackfan, K. D., and Baty, J. M.: Erythroblastosis Fetalis and Its Association with Universal Edema of the Fetus: Icterus Gravis Neonatorum and Anemia of the Newborn, J. Pediat. **1**:269, 1932.

3. Zimmerman, H. M., and Yannet, H.: Kernicterus: Jaundice of the Nuclear Masses of the Brain, Am. J. Dis. Child. **45**:740 (April) 1933.

and cerebellar cortex as well as in the grossly icteric basal ganglions. And they stated, "An appreciable percentage of cases that reveal the symptom complex of icterus gravis present evidence that may be taken as indicative of involvement of the central nervous system (convulsions, spasticity, irregular respirations, drowsiness, vomiting); of these only a small percentage show kernicterus post mortem."

The same two authors [4] in 1935 reported on a child with a stormy icteric course at birth who subsequently lived to the age of 3, showing during life signs of diffuse involvement of the central nervous system associated with athetotic movements of the extremities. Microscopic examination of the brain at autopsy showed changes strikingly similar to those of kernicterus, though nuclear staining had not been seen in the gross examination. They again put forward the concept of the clinical diagnosis of kernicterus. De Lange [5] in 1935 showed the lack of relationship between the degree of kernicterus and the amount of damage seen on microscopic section. Levine and Stetson [6] in 1937 implicated an isoimmunization factor in the pathogenesis of erythroblastosis, later proved to be the Rh factor by Landsteiner and Weiner [7]; and with this development began what might be called the modern concept of the disease and its present day therapy. Sobel and Zucker [8] in 1940 reported on a case in which a patient at 3½ years with neuromuscular defects, who had previously had erythroblastosis, died and did not show the usual changes of kernicterus at autopsy, but showed "diffuse changes of a moderate degree scattered throughout the cerebral hemispheres and diencephalon." Leonard [9] in 1945 reviewed 55 cases of erythroblastosis at New York Hospital for the years 1938 to 1944. Nineteen of the patients died in the neonatal period, 5 of them showing kernicterus at autopsy. Of the surviving 36, 1 shows spasticity and mental retardation attributed to kernicterus. On the other hand, Docter [10] in 1945 reported that of 13 patients with erythro-

4. Zimmerman, H. M., and Yannet, H.: Cerebral Sequelae of Icterus Gravis Neonatorum and Their Relation to Kernicterus, Am. J. Dis. Child. **49**:418 (Feb.), 1935.

5. de Lange, C.: Nuclear Icterus With and Without Erythroblastosis, Jahrb. f. Kinderh. **145**:273, 1935.

6. Levine, P., and Stetson, R.: An Unusual Case of Intra-Group Agglutination, J. A. M. A. **113**:126 (July 8) 1937.

7. Landsteiner, K., and Weiner, A. S.: An Agglutinable Factor in Human Blood Recognized by Immune Sera for Rhesus Blood, Proc. Soc. Exper. Biol. & Med. **43**:223, 1940.

8. Sobel, I. P., and Zucker, J. M.: Icterus Gravis Neonatorum: End Results of the Syndrome of Nuclear Jaundice, J. Pediat. **16**:445, 1940.

9. Leonard, M. F.: Hemolytic Disease of the Newborn: Clinical Analysis of Fifty-Five Cases with Special Reference to Pathogenesis; Prognosis and Therapy, J. Pediat. **27**:249, 1945.

10. Docter, J. M.: Kernicterus: Neurological Sequelae of Erythroblastosis Fetalis, J. Pediat. **27**:327, 1945.

blastosis that survived the neonatal period, 5, or 38 per cent, showed manifestations of kernicterus, while 6 of the 7 that died showed this lesion at autopsy.

As to the pathogenesis of kernicterus, recently there has been postulated what might be considered a modern version of one of the possibilities suggested by Bencke in 1907. In a recent article, Weiner [11] has suggested that the agglutinated red cells plug the capillaries of the brain, resulting in anoxemia. The ganglion cells, being most susceptible to short periods of anoxia, die and are then stained by the bile in circulation. This leaves a flexible enough concept to explain the occurrence of degeneration in other portions of the brain, making it a circumstance dependent on the location of the emboli.

The paper by Docter and 1 of the cases to be presented here (D. J.) prompted me to undertake a like survey of all proved cases of erythroblastosis fetalis in the records of Children's Hospital, with a view to ascertaining the number of living kernicteric children in this group.

METHOD

In any study of this sort, the problem invariably occurs which cases to include and which to discard in a series. Culling through all the records in Children's Hospital from 1920 up to and including Dec. 31, 1945, there were 47 cases that could reasonably be thought to fit the pattern of erythroblastosis. In most there was a presenting complaint of jaundice in the first few days of life, but in some merely one of progressive anemia. Not having an obstetric service, the Children's Hospital of necessity received only patients that were considered too ill to be treated in the surrounding hospitals; consequently most of the children showed a rather clearcut pattern of erythroblastosis. However, many of those prior to 1943 had had no Rh determinations made. Therefore an attempt was made to have the mother and child in these families typed and to ascertain the child's developmental status. Although 10 per cent of patients with erythroblastosis do not fit the Rh pattern, it was felt desirable to omit all such patients from this study, as well as to omit all for whom an adequate follow-up was not obtainable. My criteria, therefore, for inclusion in this series are the typical pattern of an Rh-positive child born of an Rh-negative mother, with the presenting complaint of either neonatal jaundice or anemia.

COMMENT AND REPORT OF CASES

There were 35 patients who fitted these criteria for whom either a clinical follow-up or an autopsy was obtained. Of these 35, 4 were Negro and the rest were white. Six of the 35 have died, and it might

11. Weiner, A. S., and Brody, M.: The Pathogenesis of Kernicterus, Science **103**:570, 1946.

be of interest to look at these 6 before passing on to the 29 living with whom this paper is more directly concerned. Only 2 of these 6 showed any cerebral involvement that might be construed as being related to kernicterus. One, a white child, 4 days old, jaundiced since birth, had a palpable liver and a red cell count of 1,900,000. There were 4,000 nucleated red cells per cubic millimeter. The child had several convulsions and died on the day of admission. At autopsy the liver and spleen were enlarged, showing congestion on microscopic section. There were areas of yellow pigmentation scattered throughout the cortex and the brain stem. On fixation, yellow and yellow-green pigment was seen chiefly in the brain stem. Staining of the basal ganglions was not noted.

The second of these 2 was a premature Negro infant weighing 4 pounds (1,814 Gm.), who became jaundiced on the third day and died on the sixth. The red blood cell count was 2,900,000. At autopsy, the significant findings included enlargement of the liver with areas of hemopoiesis and a large congested spleen. There were areas of localized pigmentation in the floor of the fourth ventricle and insula.

Three of the remaining 4 who died showed no abnormalities of the brain at autopsy. In the last case of the 6 the brain was not examined. It is of interest that 1 of these 3 with no pathologic cerebral conditions showed clinical involvement of the central nervous system. This was a white child, whose jaundice appeared on the second day of life and became rather intense, registering an icteric index of 120. There were 7 previous siblings most of whom were jaundiced at birth, but no detailed history as to severity was obtainable. All 7 were living and well. The liver and the spleen were both enlarged in this child, and there were 2,700,000 red blood cells. On the third day the child's respirations became gasping in type, and it took an opisthotonic position. In this condition it left the hospital after a forty-three day stay, having received four transfusions totaling 320 cc. of blood, some of which may not have been Rh negative. It returned about two weeks later, marasmic in appearance, with thrush. It showed opisthotonos, was spastic on the right and held its mouth open with deviation of the jaw to the right. At autopsy the brain was grossly normal, and sections were reported as showing no signs of kernicterus. There was a generalized engorgement of the cerebral vessels.

These 6 deaths bear out the literature in the lack of correlation between the clinical and the pathologic picture, an observation that has been noted before by Zimmerman and Yannet [3] and de Lange.[5] That none of the cases with evidence of pigmentation of the central nervous system was typical of kernicterus in its classical form is not discouraging, as there is evidence in the literature that the damage need not be in the basal ganglions (de Lange [5] and Sobel [8]).

Twenty-nine of the 35 patients in this series are living. Of these, 4 have failed to develop normally. There is no history of trauma at birth in any of these 4, and in the presence of the criteria for erythroblastosis fetalis it is felt that these patients represent a group of surviving kernicteric children. The age of the children included in this group of 29 may be seen in table 2. All 4 of the defective children were recognizably backward before the age of 1 year, 2 of them showing signs in the neonatal period. Only 10 of this series were, when last observed, under the age of 1 year, and it appears highly unlikely that any of the children reported here as developing normally will, as time goes on, show defects attributable to their erythroblastosis. It is of interest that of the 4 Negro children reported on in this series, 3 showed involvement of the central nervous system. Two of them are in the living series, and 1 among those that came to autopsy showed some cerebral pigmentation.

Case 1.—D. J., a 4 day old white girl, was admitted to Children's Hospital with a history of jaundice since birth. The infant was full term, apparently the product of a normal labor and delivery. She was given 60 cc. of plasma intramuscularly and fluids subcutaneously at another institution. She did not take her formula well, and one day before admission she vomited some fresh blood. Physical examination on admission revealed an intensely jaundiced 4 day old mildly dehydrated infant, lying in partial opisthotonos. She had a weak, feeble cry, and the temperature was 102 F. There was a moderate degree of cyanosis. Respirations were irregular, with prolonged periods of apnea. The fontanelle was not bulging, and there were no meningeal signs. The tip of the spleen was barely palpable, and the liver 2 fingerbreadths below the costal margin. The remainder of the physical examination revealed no abnormality.

The mother had had 1 previous pregnancy that ended in a 6 month nonviable premature infant. Rh determinations were done and revealed that the patient and father were Rh positive, the mother Rh negative.

Laboratory data on admission revealed a negative Kahn reaction of blood serum and spinal fluid. Initial blood cell count showed 9.5 Gm. of hemoglobin, 3,200,000 erythrocytes and 12,600 leukocytes, with a normal differential count. An occasional nucleated red cell was seen. Other laboratory findings included an immediate direct van den Bergh reaction and a bilirubin content of 6.2 mg. per hundred cubic centimeters. This later dropped to 2.4 mg. per hundred cubic centimeters three weeks after admission. Nonprotein nitrogen in the blood was 36 mg. per hundred cubic centimeters, phosphorus was 4.4 mg. and calcium was 12.4 mg. Prothrombin time was 79 per cent of normal. Urine showed initially 50 mg. of albumin per hundred cubic centimeters and some white blood cells. The stool constantly contained bile.

Roentgenograms of the chest and the long bones did not indicate any pathologic condition.

During a hospital stay of forty-two days, the child's condition gradually improved under a regimen that consisted of oxygen, respiratory stimulants, chemotherapy and transfusions (a total of two transfusions, 140 cc. of Rh-negative blood). The temperature returned to normal on the fifth hospital day and stayed there, although the urine continued to show numerous white blood cells in clumps with albumin values up to 100 mg. for some time after the patient became afebrile.

TABLE 1.—*Data on Thirty-Five Patients with Erythroblastosis Fetalis*

Case Number	Patient	Race *	Siblings	Jaundice	Anemia †	Enlargement of Liver	Enlargement of Spleen	Neurologic Manifestations ‡	Outcome
1	D. J.	.....	1 dead after 6 mo.' pregnancy	+	Moderate	+	—§	(1) Flaccid extremities; (2) opisthotonos; (3) apnea and cyanosis	Kernicterus
2	A. P.	Negro	2 living and well	+	Severe	+	+	—	Kernicterus
3	C. S.	.....	2 living and well	+	Mild	+	—	Spasticity of arms	Kernicterus
4	M. L.	Negro	8 living and well; 1 (younger) erythroblastotic	+	—	—	—	—	Kernicterus
5	J. O.	.....	?	+	Severe	+	—	Convulsions	Died; pathologic condition of brain
6	D. P.	Negro	1 living and well	+	Moderate	—	—	—	Died; pathologic condition of brain
7	B. B.	.....	1 living and well	—	Moderate	+	+	—	Died
8	J. H.	.....	7 living and well; some jaundiced at birth	+	Moderate	+	+	(1) Gasping respirations; (2) opisthotonos	Died
9	B. L.	.....	2 living and well; in 1 spontaneous abortion	—	Severe	—	—	—	Died
10	R. D.	.....	4 living and well	—	Severe	+	+	—	Died
11	P. H.	.....	0	+	Moderate	—	—	—	Well
12	J. F.	.....	Premature twins died 8 hours after birth	—	Severe	+	—	—	Well
13	W. P.	.....	?	—	Moderate	—	—	—	Well
14	J. M.	.....	1 living and well	+	Moderate	+	+	—	Well
15	J. D.	.....	0	+	Mild	+	+	—	Well
16	D. C.	.....	1 jaundiced at birth	+	Mild	—	—	—	Well

17	M. O.	.....	2 living and well	—	Moderate	—	—	—	Well
18	J. M.	.....	0	+	Moderate	—	—	—	Well
19	K. S.	.....	1 spontaneous abortion (3d mo.); 1 premature infant (8th mo.), who died	+	Moderate	+	+	—	Well
20	B. B.	.....	1 living and well	+	Moderate	+	+	—	Well
21	B. L.	.....	2 living and well	+	Moderate	—	—	—	Well
22	B. D.	.....	4 spontaneous abortions	+	—	—	—	—	Well
23	B. Mc.	.....	3 normal; a fourth died 5 days post partum, jaundiced	+	Moderate	+	+	—	Well
24	A. D.	.....	1 living and well; 1 abortion (5½ mo.)	+	Moderate	—	—	—	Well
25	B. G.	.....	2 living and well	+	Moderate	+	+	—	Well
26	B. C.	.....	3 spontaneous abortions	+	Moderate	+	+	—	Well
27	G. C.	.....	2 living and well	+	Moderate	+	+	—	Well
28	J. H.	.....	1 normal, 5 jaundiced; twin of patient died, jaundiced	+	Severe	+	+	—	Well
29	M. S.	.....	0	—	Moderate	—	—	—	Well
30	J. R.	.....	1 living and well	+	Severe	—	—	—	Well
31	B. D.	.....	3 living and well	+	Moderate	+	—	—	Well
32	B. B.	.....	0	+	Severe	+	—	—	Well
33	T. S.	Negro	3 living and well	+	Severe	+	—	—	Well
34	B. S.	.....	1 living and well	+	Severe	+	+	—	Well
35	V. T.	.....	1 living and well	+	Moderate	—	—	—	Well

* All patients were white except patients whose race is specified as Negro.

† Anemia is called mild when there are 4,000,000 to 5,000,000 erythrocytes, moderate when there are 2,000,000 to 4,000,000 and severe when there are less than 2,000,000.

‡ These manifestations occurred in the first week of life.

§ The dash indicates that either the condition listed was absent or there was no record of it on the chart.

Culture of urine showed Escherichia coli. The hemoglobin ranged from the low point found on admission to over 20 Gm., and there were over 5,000,000 red blood cells soon after admission. This was followed by a gradual subsidence to lower levels. Clinically, the jaundice slowly waned. Respirations gradually improved but never became completely regular. Some spasticity of the extremities was noted during the child's hospital stay. In this condition she was discharged from the hospital with a diagnosis of erythroblastosis fetalis, pyelitis and possible kernicterus.

For the next three months she was seen several times in the dispensary, usually for infections of the upper respiratory tract. There are several scattered notes concerning the child's lethargy, and bilateral strabismus was noticed by one examiner.

At the age of 5 months, the infant was brought to the dispensary with a history of progressive opisthotonos and spasticity of two and one-half weeks' duration. Examination at this time revealed, in addition, an absence of light perception and sunken, internally strabismic eyes.

On June 13, 1946, at the age of 6 months, the child was readmitted to the hospital because of a temperature of 105 F. associated with listlessness and

TABLE 2.—*Present Age of Living Patients with Erythroblastosis Fetalis*

	Age					
	Birth to 6 Mo.	6 Mo. to 1 Yr.	1 Yr. to 18 Mo.	18 Mo. to 2 Yr.	2 Yr. to 6 Yr.	Total
Well children	3	7	4	6	5	25
Children with kernicterus	1	0	0	2	1	4
Totals	4	7	4	8	6	29

stertorous breathing. Physical examination revealed a dehydrated, critically ill white girl, with nuchal rigidity, rapid respirations and a chest filled with numerous coarse and fine moist inspiratory rales. A spinal tap yielded clear fluid under no increase in pressure. Subsequent analysis gave negative results. The red blood cell count was 3,900,000, with 12.5 Gm. of hemoglobin and 19,700 leukocytes, of which 48 per cent were polymorphonuclear. The urine initially showed 40 mg. of albumin per hundred cubic centimeters, though subsequently there was none.

The child recovered from its acute infection on a regimen that included sulfadiazine, penicillin and oxygen. However, it continually had trouble with its feedings, aspirating food easily and had transient attacks of cyanosis. There was no improvement in its neurologic status, which remained substantially as it was prior to this acute episode. It was noted that the spasticity was changeable in type, sometimes being more marked in one limb, sometimes in another. Gentle pressure was all that was required to break the spasticity, and it was felt that the picture was more in keeping with disease of the basal ganglions than with involvement of cortex or internal capsule. The cranial nerves were intact except for an internal strabismus bilaterally. The fundi showed snowy white disks. Reflexes were hyperactive and gross sensation response was delayed. It was felt that this neurologic picture could be explained by kernicterus.

CASE 2.—A. P. was brought to Children's Hospital at the age of 12 days with a referral note from another hospital stating that she had been jaundiced since birth. She had had a transfusion of 85 cc. of blood in the other institution. The mother's delivery had been normal. There were 2 siblings living and well.

On admission the infant was severely icteric, appearing dull and somewhat toxic. The posterior fontanelle was almost closed, while the anterior was open 2 fingerbreadths. The abdomen was slightly distended, the liver and spleen palpable 3 fingerbreadths below the costal margin. Laboratory studies revealed that the mother was Rh negative and the child Rh positive. Erythrocytes were 2,700,000 and hemoglobin 8.5 Gm. An occasional nucleated red cell was seen on the initial smear. With transfusion therapy, a total of 170 cc. on two separate occasions, the count rose to 4,600,000 red blood cells and 15 Gm. of hemoglobin during her hospital stay. Other pertinent laboratory findings included an icteric index of 132 units. The van den Bergh reaction was immediate direct; there was 14 mg. of bilirubin per hundred cubic centimeters, and the Kahn reaction was negative. The prothrombin time was 80 per cent of normal. The urine was not remarkable except for a positive reaction for bile. Roentgen examination of the long bones showed no pathologic conditions.

The child made an uneventful recovery and with the clearing of the jaundice was discharged after a hospital stay of thirty-one days.

In the months following discharge, the infant was seen in the dispensary at varying times for infections of the upper respiratory tract. At the age of 5 months it was noticed that the anterior fontanelle was bulging slightly, that there was no control of the external ocular muscles and that there was slight spasticity of the right arm. The child was seen in the neurologic clinic at the age of 6 months, and the following note was made: "She will follow mother with her eyes, not able to hold head up, shows no interest in surroundings, does not play with rattle and moves all extremities well. Dentition is at 5 months." The cranial nerves were intact except for paresis of the right sixth nerve. Tonus was judged to be good. Reflexes were present and equal.

At the age of 17 months she was readmitted to the hospital for a pneumoencephalogram. Physical examination at this admission revealed a quiet child who did not protest manipulation and was unable either to sit up or to hold its head up. Laboratory tests showed a moderate degree of anemia. No abnormality was noted at any urinalysis. Examination of the spinal fluid showed 5 mg. of protein and 25 mg. of sugar per hundred cubic centimeters and 10 leukocytes per cubic millimeter, of which 90 per cent were lymphocytes. Reactions to colloidal gold and Wassermann tests were negative. The pneumoencephalogram was interpreted as showing the ventricular system to be well outlined and dilated throughout, particularly in the third ventricle. There was excessive air over the frontal cortex, possibly indicative of either degeneration or agenesis. She was discharged to be followed in the neurologic clinic.

At the age of 19 months she could move all extremities and was making unsuccessful attempts to sit up. She was making no attempt to speak.

CASE 3.—C. S., a 4 day old infant, was admitted to Children's Hospital with a history of jaundice appearing one day after birth. The child was full term, and delivery was normal. The infant had 2 siblings, both normal and not jaundiced at birth.

Physical examination revealed a markedly jaundiced, well developed infant boy. The edge of the liver was palpable 2½ fingerbreadths below the costal margin. The spleen was not felt. There were some impetiginous lesions in both axillas and behind the ears. Other observations were essentially negative. Rh determinations done on the parents and child revealed an Rh-positive father and child and an Rh-negative mother. Anti-Rh agglutinins were present in the mother's serum in a dilution of 1 to 128. Laboratory observations after two transfusions of 75 cc.

each (Rh-negative blood) revealed 14 Gm. of hemoglobin and 2,000,000 erythrocytes. A rare nucleated blood cell was seen on the slide.

The child recovered from the jaundice and left the hospital after a ten day stay. One examiner on two separate occasions noticed some spasticity of the arms.

The follow-up in this case has of necessity been second hand. However, by telephone conversation with the mother and by personal conversation with the child's pediatrician, it has been ascertained that at the age of 16 months he has typical spastic paraplegia, being unable to move about and showing no mental development.

CASE 4.—M. L. at the age of 5 days was brought to the dispensary of Children's Hospital with a history of sudden onset of jaundice that morning. He was the ninth child of a family of 9, the product of a normal labor. The 8 previous siblings were alive and well. The patient was not very ill and was followed in the dispensary for five days. The Kahn reaction of the blood was negative, there was 14.4 mg. of bilirubin per hundred cubic centimeters (normal 0.1 to 0.5 mg.) and the icterus index was 100 units. The hemoglobin was 11 Gm. and erythrocytes 2,600,000. There was a normal differential count. No nucleated red cells were seen.

Fifteen months later the child was admitted to the hospital for an infection of the upper respiratory tract. At this time it was noted that he made numerous purposeless movements of his extremities. He was unable to sit up or walk unaided. Various observers described his expression as "blank," although there was some comment concerning his response to being played with and his ability to recognize and reach for toys. Several different examiners agreed in essence on the presence of choreiform movements and the absence of any other significant neurologic findings. With the exception of some mild respiratory infection the remainder of the physical examination revealed no abnormality. Laboratory studies showed the urine to be normal; there was a moderate degree of anemia and the spinal fluid showed no abnormalities. A pneumoencephalogram showed a considerable increase of air in the sulci that was interpreted as possibly indicating some cortical atrophy.

Rh determinations were made at this time. The patient and his father were Rh-positive, the mother Rh-negative. Subsequent investigation revealed that with the birth of her eighth normal child there was some postpartum bleeding and that the mother received several transfusions, most of which were undoubtedly with Rh-positive blood, as this occurred in 1940. The patient was the first offspring following this incident, and another child born subsequent to this patient, the mother's tenth, was jaundiced at birth. The patient was discharged from the hospital with the diagnosis of kernicterus.

At present, at the age of 3 years, he can walk with a peculiar ataxic lurching gait. He makes continuous purposeless movements with his extremities and head. In reaching for objects he overshoots, then recovers and manages to grasp what he is after. He appears to be mentally alert, can say about ten words and gets on well with his siblings.

There appears to be no correlation in these cases with regard to the degree of jaundice, the intensity of the anemia, the number of transfusions given and the degree of enlargement of the spleen and the liver. As is to be expected, none of these children were first born. Two of them had 2 elder siblings, and 1 had 1. In 2 cases these were normal children, and in 1 case (D. J.), death was by spontaneous abortion in

the sixth month. Of interest is case 4, in which, after 8 previous normal births, the last of which necessitated multiple transfusions of Rh-positive blood, the patient was born; an ensuing child was also erythroblastotic. The family history of this case has been reported previously by Relfe and Adams.[12] Of the 4 cases presented here, 2 showed signs of involvement of the central nervous system in the neonatal period. None of the 25 normal children showed any such involvement. Four out of 29 is not a large enough series from which to draw dogmatic conclusions, but it appears to be not too rash a statement to say that the prognosis must be guarded whenever involvement of the central nervous system, no matter how mild, occurs in a newborn infant with erythroblastosis.

In case 2 (A. P.) there was cortical atrophy in the pneumoencephalogram. Again, in this case, the argument might be advanced that this need not be kernicterus. This is admitted, but in view of the possibility that damage to the brain from erythroblastosis is diffuse and not limited to the basal ganglions, it cannot be disproved. Weiner [11] has stated that "infants with kernicterus occasionally survive, in which case they develop signs of a diffuse neurological disorder of varying severity, usually accompanied by mental deficiency." Unfortunately, no statistics appear to be available for comparison on the incidence of neuromuscular defects in any consecutive series of normal births. It is apparent, however, that 4 out of 29, or 15 per cent, is a significant figure.

In the light of these figures, the outlook for an erythroblastotic child is not nearly so hopeless as was implied by Docter. The great majority of those that survive will go on to develop normally. Whether transfusion therapy of an intensive nature should be used for those infants that show some involvement of the central nervous system at birth is another matter, but obviously, in the light of present knowledge, such treatment could not be withheld, as the definite prognosis for these children is not yet established. It might be well to point out that transfusion therapy is merely substitution therapy, and that in the presence of pronounced damage to the central nervous system death may still supervene in the presence of a normal hemogram. Weiner [13] stated that transfusion therapy in erythroblastosis should not be used indiscriminately, but should be reserved for those that show a low blood cell count, as it will have no salutary effect and may well be detrimental to those with a normal or high red cell count.

12. Relfe, J. D., and Adams, J. C.: Erythroblastosis Fetalis, Clin. Proc. Child. Hosp. **1**:210, 1945.

13. Weiner, A. S., and Sonn, E.: Pathogenesis of Congenital Hemolytic Disease, Am. J. Dis. Child. **71**:25 (Jan.) 1946.

SUMMARY AND CONCLUSIONS

Of 35 patients with erythroblastosis for whom follow-up records were obtainable, 29 are still living and 6 have died. The ratio of white to Negro in this series was 31 to 4.

Analysis of the autopsy reports of the 6 deaths failed to show any correlation between the degree of clinical involvement of the central nervous system and the pathologic picture.

Of the 29 living children 4, or 15 per cent, show signs of involvement of the central nervous system that may be interpreted as evidence of kernicterus. Two of these 4 showed evidence of involvement of the central nervous system in the neonatal period. None of the remaining 25 normal children showed any such neonatal involvement.

Transfusion therapy should be directed against the anemia and is of no value in ameliorating the damage to the brain that has already occurred.

MANAGEMENT OF WHOOPING COUGH WITH SPECIAL REFERENCE TO INFANTS

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THERE were 3,668 recorded deaths from whooping cough in the United States in 1941.[1] Of these, 2,572 occurred in children under 1 year, and 1,048 between 1 and 3 years. Ninety-four per cent of the deaths, therefore, were in children less than 3 years old. The incidence of the disease in the Greater New York area has been fairly constant over a period of years, about 5,700 cases being reported by the New York City Health Department yearly in the years 1940 to 1945 inclusive, with a yearly average of 45 deaths. Sixty-seven per cent of these deaths occurred in children under 1 year of age (table 1).

TABLE 1.—*The Incidence and Mortality of Whooping Cough as Reported by the Department of Health of New York City*

Year	Number of Cases		Deaths	
	Total	Under 1 Yr.	Total	Under 1 Yr.
1940	5,775	429	49	38
1941	7,810	864	45	35
1942	9,240	1,075	58	42
1943	3,678	463	43	30
1944	2,810	420	33	22
1945	4,990	760	35	26

In the past eight years the case fatality for patients ill with whooping cough at the Willard Parker Hospital has been equal to or above the combined total deaths due to measles, scarlet fever and diphtheria. The Willard Parker Hospital is the only hospital for communicable diseases in the boroughs of Manhattan and Bronx. Therefore it receives most of the seriously ill patients with whooping cough in these boroughs. Since there is little or no immunity to whooping cough at birth, an appreciable percentage of these sick patients are young infants, who often come from families of low economic status. They are sometimes admitted exhausted, malnourished and in poor general physical condition. Of the 3,843

From the Willard Parker Hospital, Department of Hospitals, New York City.

1. Vital Statistics of the United States, 1941, United States Department of Commerce, Bureau of the Census, 1943, pt. 1, pp. 106-112.

patients treated at Willard Parker Hospital for whooping cough during the years of 1940 to 1945 inclusive, 475 were infants under 6 months of age and 412 were between 6 and 12 months old (table 2).

The purpose of this paper is to describe the treatment of these 887 infants under 1 year of age. Over a period of years the medical and nursing staffs at the hospital have employed a number of procedures which have had a favorable influence on the clinical course of the disease in these young infants.

Because of the well known difficulty in establishing a diagnosis of whooping cough before the occurrence of the typical whoop, only a small number of children (2 per cent of our series) were admitted before the tenth day after the cough was first noted. About 77 per cent were

TABLE 2.—*The Incidence and Mortality of Whooping Cough at the Willard Parker Hospital*

Age	1940			1941			1942		
	No. of Patients	Deaths	Mortality, %	No. of Patients	Deaths	Mortality, %	No. of Patients	Deaths	Mortality, %
0-6 mo.	47	6	12.0	50	3	6.0	128	10	8.0
6-12 mo.	42	..	....	51	2	4.0	127	3	2.4
1-2 yr.	74	..	....	138	1	0.7	244	3	1.2
2+ yr.	266	2	0.7	359	2	0.77	576	..	...
	429	8	1.8	598	8	1.3	1,075	16	1.4
	1943			1944			1945		
0-6 mo.	64	4	6.3	55	1	1.8	121	6	5.0
6-12 mo.	50	3	6.0	49	4	8.1	103	2	2.0
1-2 yr.	130	3	2.3	105	1	0.9	191	4	2.1
2+ yr.	229	3	1.3	217	1	0.5	443	1	0.2
	463	13	2.8	420	7	1.6	858	13	1.5

admitted from ten to sixteen days after the onset of cough and the remainder, 21 per cent, later than the sixteenth day.

In early cases, if the diagnosis was suspected, it sometimes could be made bacteriologically by means of a cough plate or culture taken from the nasopharynx. We found the nasopharyngeal method of culture more satisfactory than the cough plate method. The culture was taken by passing a fine metal applicator with a sterile cotton swab into the nasopharynx, as described by Brooks, Bradford and Berry.[2] In growing this culture the method employed was that described by Bullowa, Buxbaum and Scheinblum.[3] In a recent series, positive cultures were obtained for 80 per cent of the children sometime during their illness.

2. Brooks, R.; Bradford, W. L., and Berry, G. P.: The Method of Nasopharyngeal Cultures in the Diagnosis of Whooping Cough, J. A. M. A. **120**:883 (Nov 21) 1942.

3. Bullowa, J. G. M.; Buxbaum, L., and Scheinblum, I. E.: Pertussis: Bacteriologic and Agglutination Studies, J. Pediat. **25**:299 (Oct.) 1944.

Blood counts were often helpful in establishing a diagnosis. Some children had a total white blood cell count of 100,000 or over, and there was 1 child whose total leukocyte count was 240,000. A high cell count had no prognostic significance.

Roentgenograms of the chest were made if there was any question of pneumonia or atelectasis. Roentgenograms, taken in the lateral and oblique positions, at times gave valuable information.[4]

The clinical picture of an infant seriously ill with whooping cough during the paroxysmal stage is well known. There are frequent exhausting paroxysms and moderate cyanosis. The infant sleeps little, vomits frequently and becomes exhausted. At this stage of the illness the trachea, the bronchi and the bronchioles frequently contain a sticky, tenacious, viscous secretion which often becomes inspissated. Older children are able to cough up these tenacious plugs after a severe paroxysm. Young exhausted infants, however, may not have the power to expel this secretion.

METHOD OF TREATMENT

One or all of the following were essential in the care of the infant ill with whooping cough: (1) an experienced medical and nursing personnel, (2) the use of oxygen, (3) aspiration of the upper respiratory tract, (4) chemotherapy, (5) hydration and the use of parenteral therapy, (6) special feeding technic and (7) the use of hyperimmune and rabbit serums. The efficacy of these serums will be discussed in a subsequent publication.

1. *Nursing.*—It is axiomatic that good nursing is essential in the recovery of any seriously ill infant. This is especially true in whooping cough in which the use of all of the technics just listed may be required. The seriously ill infant never should be left unattended, for the onset of a paroxysm may be sudden and unexpected. The nurse must be schooled in the use of all mechanical life-saving measures, including the insertion of an airway, the use of suction and oxygen and the mechanics of the oxygen chamber. Prompt and correct judgment as to procedure can be a life-saving measure, especially in infants.

2. *Oxygen Therapy.*—All severely ill infants and young children have varying degrees of anoxemia. This anoxemia is due mainly to (1) the character of the mucoid secretion plugging the bronchial tree and causing varying degrees of atelectasis and pulmonary infiltration; (2) extensive pulmonary involvement in which excessive mucous secretion does not seem to be important, and (3) frequent long paroxysms with or without mucus or obstruction in the trachea and the bronchi.

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4. Kohn, J. L.; Schwartz, I.; Greenbaum, J., and Daly, M. M. I.: Roentgenograms of the Chest Taken During Pertussis, Am. J. Dis. Child. **67**:463 (June) 1944.

It has been our practice to place every young infant, on admission, in an oxygen tent for a trial period. The duration of treatment is determined by the general condition of the child, and also by the clinical evidence of extensive pneumonia and atelectasis. The period of oxygen therapy varies from tweny-four hours to six weeks.

Children who had exhausting paroxysms without evidence of pneumonic infiltration were often relieved when placed in an oxygen tent. The atmosphere of the oxygen tent acted as a sedative, the child often fell asleep, and the paroxysms became less prolonged and exhausting.

Oxygen therapy also proved to be a valuable aid in those children who had convulsions. Many such patients were relieved quickly when placed in the oxygen tent and were kept there until there was no further evidence of cerebral irritability. The removal of some spinal fluid through lumbar puncture was tried, but it usually was ineffective in relieving this symptom. Occasionally, on recovery, such infants showed evidence of permanent cerebral injury such as spasticity of the extremities, hemiplegia or mental retardation. Dolgopol [5] has described the changes which occur in the brain of children who have had convulsions and anoxia.

The temperature of the tent was kept at about 68 F. and the humidity close to 40 per cent. The oxygen concentration was about 50 per cent. Lower temperatures seemed to stimulate respiration and increased the number of paroxysms. A tent having a transparent canopy was preferable to one with a rubberized fabric and enclosed windows. The oxygen tent was found superior to facial masks or nasal catheters, since these devices often frightened the child. They were used only in emergencies when an oxygen tent was not available immediately.

3. *Aspiration.*—As previously noted, about two weeks after the onset of whooping cough the trachea and bronchi often contained a thick tenacious secretion which the exhausted infant was unable to expel because of the small caliber of the respiratory passages. Suction was applied to the pharynx and trachea of every infant with any signs of obstruction. It must be appreciated that in order for the oxygen to reach the bronchial tree, an attempt must be made to clear the air passages. Frequent change of the infant's position in the crib was helpful in preventing stagnation of the secretions.

For severely ill infants, a standard electrically driven aspiration apparatus was kept at the bedside. For the aspiration a soft rubber catheter with multiple perforations for about 2 inches (5.1 cm.) above the tip was used. The tip was not perforated. A number 12 French-sized catheter was employed for the oral route and a number 10 catheter for the nasal route.

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5. Dolgopol, V.: Changes in the Brain in Pertussis with Convulsions, Arch. Neurol. & Psychiat. **46**:477 (Sept.) 1944.

During a severe paroxysm the child was placed in the sitting position with the head held forward, the forehead being supported. Pharyngeal suction was then applied. This was repeated as often as necessary. If a suction apparatus was not immediately available, suspension of the infant by the legs for a short period often gave temporary relief. Much exudate drained from the nose and mouth when the patient was held in that position.

Occasionally the obstruction was so complete that the infant stopped breathing. When this occurred, the child was immediately removed from the tent and placed on a stretcher or table and the larynx and trachea were aspirated. Manual resuscitation by means of the pressure and release method was employed by another person. During this procedure, oxygen or oxygen with 5 per cent carbon dioxide was administered through a funnel kept close to the mouth. When the child again breathed regularly he was returned to the tent. After such procedures the child was under continued surveillance. Some of these patients later showed evidence of cerebral injury, presumably as a result of anoxia.

4. *Medicinal Therapy.*—It has been our impression that the time-honored cough mixtures which contained expectorants and antispasmodics were of limited value in the relief of symptoms. We have not utilized any of these mixtures for a number of years. Sedatives and morphine derivatives were seldom used. Occasionally one of the barbiturates was given.

Sulfapyridine, sulfathiazole and later sulfadiazine have been prescribed for the patients reported on in the present study. The rationale of their use was the control of secondary infection. However, neither sulfonamide compounds nor penicillin had any bacterial influence on Hemophilus pertussis. Sulfapyridine, used early in this series of cases, while effective in reducing fever, produced leukopenia in some patients. Sulfathiazole was fairly satisfactory in controlling secondary infection, but it induced vomiting, which was undesirable in whooping cough. Sulfadiazine, now in general use, was the most effective of the sulfonamide drugs. In most instances after its use the temperature became normal in from one to three days. However, the clinical symptoms and signs of pneumonia sometimes persisted. It was our impression that when a mixed infection with H. pertussis and one of the pyogenic cocci was present, considerable improvement could be expected. On the other hand, it was known that extensive pneumonia with whooping cough might be caused solely by H. pertussis as shown by postmortem examination of the lungs. After sulfonamide therapy was stopped in some instances, fever returned. Nevertheless, we felt that sulfonamide compounds still were valuable in treatment. If, after five or six days, however, there was no apparent improvement, we expected no further benefit from the use of the drug.

Penicillin was not obtainable until the last year of this study. It was given to only 9 patients in this series. For 3 of these it was used only for a short period during an acute attack of otitis media. It was given to 6 other patients who were seriously ill and who had pneumonic infiltrations. Two of the latter children died. However, subsequent cases have given us the impression that penicillin was more effective than the sulfonamide compounds. It was less toxic and could be given for long periods without any harmful effect. When both penicillin and sulfonamide drugs were used, improvement was also noted. It was difficult to be certain which of the drugs had the more beneficial influence or whether they were more effective when used together than when either was administered separately.

5. *Parenteral Therapy.*—Many infants were admitted in a state of severe dehydration. It must be remembered that some of these infants had been vomiting for days. Therefore, as soon as practical after admissions, these children received fluids parenterally, whole blood, plasma, dextrose and isotonic solution of sodium chloride. If a child was admitted in ketosis or alkalosis, the appropriate solutions were administered until a normal chemical balance was restored.

6. *Feeding.*—We found it important to use a special technic in the feeding of severely ill infants and young children. In debilitated infants there was always danger of aspiration of food during or following a paroxysm. Infants were fed frequently and in small amounts. In those infants in whom there was evidence of accumulation of mucus in the upper respiratory tract, pharyngeal aspiration was instituted before the feeding. When the child became exhausted and had difficulty in sucking, fluids were given with a medicine dropper. Gavage feeding was administered only to extremely weak infants. This procedure should be avoided, since the passage of the tube may initiate a paroxysm followed by emesis.

The child was always fed in a recumbent position in the nurse's arms. After feeding, the infant was placed on its side and suction was applied through the nose. It was an inviolable rule that a nurse was never to leave the baby during a feeding. When a child had a severe paroxysm and vomited soon after feeding, he was placed in a sitting position and the head held forward. If there was no immediate relief, pharyngeal aspiration was used. On this feeding routine, many seriously ill children gained weight during their illness.

HISTORIES OF TWO ILLUSTRATIVE CASES

CASE 1.—S. M., a girl aged 4½ months, was admitted Nov. 11, 1944 with a history of cough of two weeks' duration. On admission the child was seriously ill and was having severe paroxysms. She had dyspnea and cyanosis and evidence of bilateral pneumonia. Soon after admission the child had a convulsion,

and the temperature reached a level of 108.6 F. (42.5 C.). She was placed in an oxygen tent and given fluids intravenously. After several hours the temperature receded somewhat. However, a temperature of 104 to 106 F. (40 to 41 C.) persisted for five days. She also received three doses of hyperimmune lyophil serum on successive days. Mucous secretions were removed frequently by aspiration. Roentgenographic examination of the chest at this time showed extensive consolidation of the upper lobe of the right lung. There was little improvement for the first four weeks. She received sulfadiazine for three weeks without apparent benefit. Then penicillin was started and was given for six days, during which period the patient began to improve. She was kept in the oxygen tent for five weeks.

The child was discharged from the hospital two months after admission. At the time of discharge a roentgenogram of the lungs showed complete resolution.

CASE 2.—P. B., an infant girl, had a history of cough for ten days. During the last three days the cough had become more frequent and was paroxysmal in character About six hours before admission to the Willard Parker Hospital, the child had a generalized convulsion of fifteen minutes' duration. There were several subsequent seizures, and the child was seen at home during one of them. She was breathing rapidly and was moderately cyanotic. Auscultation of the chest gave evidence of fluid in the trachea and in the large and small bronchi. Little air was reaching the alveoli. The child was suspended by her legs, and a large amount of thick tenacious mucoid fluid exuded from the nose and mouth. She obtained relief from this procedure, the breathing was less noisy and the cyanosis lessened.

The child was immediately transferred to the hospital. The pharynx and larynx were aspirated and the patient was placed in an oxygen tent, where she soon fell asleep. She was given fluid subcutaneously and intravenously. Pharyngeal aspiration was repeated frequently during the first forty-eight hours. Oxygen therapy was discontinued after thirty-six hours. There were no convulsions after admission. The cerebrospinal fluid showed no abnormalities.

For about twenty-four hours the patient was lethargic. This lethargy diminished slowly, and she reacted normally to her environment after five days.

The child's temperature on admission was 105 F. (40.5 C.) but fell to normal after forty-eight hours. A roentgenogram of the chest showed no abnormality. The blood count was 19,000 white cells per cubic millimeter, with 76 per cent lymphocytic cells.

The infant received 200,000 units of sodium penicillin subcutaneously daily for seven days, and three intramuscular injections of hyperimmune human pertussis serum, 20 cc. being given on alternate days after admission to the hospital.

Three weeks after admission the patient was discharged in good physical condition.

CASE FATALITY

With the method of treatment described in the paper the mortality of infants under 1 year of age has varied from 3.5 to 6.7 per cent (table 2). The lower figure was obtained in 1945 when hyperimmune serums were used extensively. The significance of this finding will be discussed in another paper.

COMMENT

We are reporting the treatment and management of 887 infants ill with whooping cough. It must be remembered that most of these infants

were seriously ill and that they often came from families of low economic status. Nearly all of them were admitted during the paroxysmal stage.

The prompt use of oxygen was an important element in the treatment. All infants severely ill from whooping cough have some degree of anoxia, due most frequently to obstruction of the respiratory tract by a thick, tenacious mucoid secretion. This results in atelectasis and pulmonary infiltration. Consequently, we feel that oxygen should be used prophylactically. Joe and Westwater [6] and Bullowa [7] were the first to appreciate the value of the oxygen tent. We have found it superior to any other form of oxygen therapy.

Drinker [8] has stressed the fact that pulmonary tissue is injured by anoxia. He stated that there were two factors which were fundamental in causing abnormal transudation into the lung: (1) anoxia and (2) consequent increased pulmonary capillary pressure, anoxia being the primary cause of increased permeability. As long as the circulation in the unaerated tissue continued, there was an increased production of tissue fluid. Therefore, the sooner oxygen therapy was given, the greater the possibility of checking the leakage in the capillaries of the lungs. A high concentration of oxygen had to be used in attempting to aerate the blood through alveolar walls often thickened by edema. It was imperative that the oxygen reach the alveoli directly.

Since the tracheobronchial tree was often obstructed by the thick mucoid secretion, it was essential to remove this obstruction so that the oxygen could reach the alveoli. The importance of tracheal and pharyngeal aspirations, whenever necessary, should be stressed.

Medicinal therapy has been disappointing. Sedatives were used infrequently and the antispasmodics and expectorants had little effect. Sulfonamide drugs and penicillin were apparently helpful in some patients.

CONCLUSION

We believe that the type of treatment here described has had a favorable influence on the course and outcome of whooping cough in infancy.

50 West Ninety-Sixth Street.

73 East Ninetieth Street.

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6. Joe, A., and Westwater, J. S.: Use of Poulton's Oxygen Tent in Bronchopneumonia Complicating Whooping Cough, Brit. M. J. **2**:292 (Aug. 17) 1935.

7. Bullowa, J. G. M.: Personal communication to the author.

8. Drinker, C. K.: Pulmonary Edema and Inflammation: An Analysis of Processes Involved in the Formation and Removal of Pulmonary Transudates and Exudates, Cambridge, Mass., Harvard University Press, 1945.

FAMILIAL HYPOPLASTIC ANEMIA OF CHILDHOOD

Report of Eight Cases in Two Families with Beneficial Effect of Splenectomy in One Case

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AMONG the anemias of infancy and childhood, those due to hypoplasia or aplasia of the bone marrow are relatively uncommon. Early reports of "aplastic" anemias are clouded in obscure and often ambiguous terminology, and frequently the designation "aplastic" is based simply on the existence of anemia, leukopenia and thrombopenia (i. e. pancytopenia) in the peripheral blood, without marrow studies to confirm the use of the term. Thus, true aplastic anemias, transient and chronic pancytopenic states associated with infection, leukemia or sarcomatous metastasis to the bone marrow and (probably) certain cases of splenic pancytopenia have often been reported under the designation "aplastic anemia."

However, a definite although small number of infants and children show anemia which is the result of true hypoplasia or aplasia of the bone marrow. Perhaps the first report was by Vaquez and Aubertin,[1] who in 1904 described a patient who had had pallor and weakness from birth, was found to have anemia at puberty, and had pancytopenia from that time until death at 19 years. The marrow at autopsy was aplastic. Kleinschmidt, in 1915,[2] described 4 children, aged 4½ to 12 years, with "hemolytic anemia with lack of marrow regeneration," in whom there was a simultaneous reduction in red cells, leukocytes, granulocytes and platelets, with no evidence of any activity of the marrow. Autopsies on 2 of the children showed a markedly hypoplastic marrow. The term "hemolytic" in the title is not substantiated. In 1919, Smith [3] collected

From the Blood Laboratory of the J. H. Pratt Diagnostic Hospital and Boston Dispensary, the Boston Floating Hospital, and Tufts College Medical School. Aided by grants from the Charlton Fund and the Upjohn Company.

1. Cited by Frank, E.: Aleukia hemorrhagica: Aplastische (aregenerative) Anämie-Panmyelophthisie: I. Aleukie und Thrombopenie, Berl. klin. Wchnschr. **52**:961, 1915.

2. Kleinschmidt, H.: Aplastische (Aregeneratorische) hämolytische Anämie im Kindesalter, Jahrb. f. Kinderh. **81**:1, 1915.

3. Smith, L. W.: Report on an Unusual Case of Aplastic Anemia, Am. J. Dis. Child. **17**:174 (March) 1919.

62 cases of aplastic anemia reported since 1888, of which 5 occurred under the age of 12; and added a sixth. Three years later, Hermann [4] listed 80 cases of aplastic anemia in the literature, of which 21 were in children and 1 in an infant. Obviously, the number of cases varied with the individual author's criteria. Baar's case of aplastic anemia in a child was first noted after infection.[5] Marquézy and Flandrin [6] recorded 2 cases of aplastic anemia, 1 of which was in a child aged 4 years. Ullrich [7] reported 5 cases of pancytopenia in children, of which 4 followed or were precipitated by infection and only 1 was an example of true idiopathic hypoplastic anemia. Hoenig [8] added a similar case.

In 1936 Josephs [9] described 2 infants with aplastic anemia whom he maintained for eighteen months and four and one-half years respectively by blood transfusions, only to have them die when the necessity for continued transfusions was ignored.[10] Kato and Steiner,[11] Peat [12] and Diamond and Blackfan [13] reported additional cases. Illing [14] described 7 children with pancytopenia, of whom the first 2 had idiopathic aplastic anemia and the other 5 pancytopenia associated with various infections. Smith,[15] Kohlbry,[16] Rinvik [17] and Rubell [18] added other instances of idiopathic aplastic anemia in childhood.

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4. Hermann, C.: Idiopathic Aplastic Anemia: Report of Case in a Child Four Years Old; Marked Improvement from Transfusion, Am. J. Dis. Child. **23**: 484 (June) 1922.

5. Baar, H.: Progressive postinfektiöse Erythroptise, Folia haemat. **35**:111, 1928.

6. Marquézy, R. A., and Flandrin, P.: Deux cas d'anémie pernicieuse aplastique: Aigue fébrile chez l'enfant, Arch. de méd. d. enf. **34**:228, 1931.

7. Ullrich, O.: Zur systemik aregeneratorischer und hyperplastischer Reaktionen des Blutsystems mit einschlägiger Kasuistik, Ztschr. f. Kinderh. **53**:487, 1932.

8. Hoenig, L.: Beitrag zur Kenntnis der aplastischen Anämie im Kindesalter, Ztschr. f. Kinderh. **53**:580, 1932.

9. Josephs, H. W.: Anemia of Infancy and Early Childhood, Medicine **15**: 308, 1936.

10. Josephs, H. W., in discussion of Diamond and Blackfan.[13]

11. Kato, K., and Steiner, P. E.: Hematology and Pathology of Aplastic Anemia Occurring in a Five Year Old Child, J. Pediat. **9**:433, 1936.

12. Peat, G. B.: A Case of Aplastic Anemia in a Child, Canad. M. A. J. **37**:64. 1937.

13. Diamond, L. K., and Blackfan, K. D.: Hypoplastic Anemia, Am. J. Dis. Child. **56**:464 (Aug.) 1938.

14. Illing, T.: Beitrag zum Krankheitsbild der Panmyelophthise im Kindesalter, Folia haemat. **62**:369, 1939.

15. Smith, C. H.: The Anemias of Early Infancy: Pathogenesis and Diagnosis, J. Pediat. **16**:375, 1940.

16. Kohlbry, C. O.: Congenital Hypoplastic Anemia, J. Pediat. **19**:662, 1941.

17. Rinvik, R.: Two Cases of Idiopathic Hypoplastic Anemia in Infants, Acta pædiat. **28**:304, 1941.

18. Rubell, I.: Hypoplastic Congenital Anemia, J. Pediat. **20**:756, 1942.

The disorder described in most of these reports, except for those with infections, was characteristically congenital. The anemia was noted early in infancy and was regularly associated with leukopenia and thrombopenia. Periodic transfusions were usually required to maintain life. Spontaneous remissions were rare.[18] Examination of the bone marrow revealed hypoplasia with little evidence of regenerative activity.

In none of these instances was a familial history of similar disorder reported. Familial hypoplastic anemia was first mentioned by Fanconi,[19] who described a fatal anemia of a peculiar type in 3 brothers aged 5, 6 and 7 years respectively. Each case showed hyperchromic anemia, leukopenia, thrombopenia, hemorrhagic diathesis, microcephaly, testicular hypoplasia, convergent strabismus, exaggerated deep tendon reflexes and generalized brown, melanin-like pigmentation of the skin but not of the buccal mucosa. The marrow was hypoplastic in the 2 cases in which this tissue was examined. Fanconi believed that this hypoplasia of the marrow was merely one of a series of constitutional defects in this family. Familial cases similar to those of Fanconi have been described by Emile-Weil [20] (2 brothers in a family of 5 children), Hjorth [21] (a brother and a sister) and Dacie and Gilpin [22] (2 brothers and a first cousin).

Sporadic cases similar to those of Fanconi have been reported by Uehlinger,[23] van Leeuwen,[24] Emile-Weil [20] and ourselves.[25] In all these cases, brownish discoloration of the skin, various congenital abnormalities (absent thumb, testicular atrophy, absent kidney, etc.) and hypoplasia of the marrow were present. The designation "Fanconi's syndrome" for these cases was used by several authors. Cases in which hypoplasia of the bone marrow was present as the only familial aberration have not previously been described.

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19. Fanconi, G.: Familiäre infantile perniziösaartige Anämie (perniziöses Blutbild und Konstitution), Jahrb. f. Kinderh. **117**:257, 1927.

20. Emile-Weil, P.: Myélose aplasique infantile familiale avec malformations et troubles endocriniens: Contribution à l'étude du syndrome de Fanconi, Sang **12**:369, 1938.

21. Hjorth, P.: Two Cases of Constitutional Anemia in Children, Nord. med. (Hospitalstid.) **7**:1313, 1940.

22. Dacie, J. V., and Gilpin, A.: Refractory Anemia (Fanconi Type): Its Incidence in Three Members of One Family with in One Case a Relationship to Chronic Hemolytic Anemia with Nocturnal Hemoglobinuria (Marchiafava-Micheli Disease or Nocturnal Hemoglobinuria), Arch. Dis. Childhood **19**:155, 1944.

23. Uehlinger, E.: Konstitutionelle infantile (perniciösaartige) Anämie, Klin. Wchnschr. **8**:1501, 1929.

24. van Leeuwen, H. C.: Ein Fall von konstitutioneller infantiler perniziösaähnlicher Anämie (Fanconi), Folia haemat. **49**:434, 1933.

25. Estren, S.; Suess, J. F., and Dameshek, W.: Congenital Hypoplastic Anemia Associated with Multiple Developmental Defects (Fanconi Syndrome), Blood **2**:85-93, 1947.

The early onset of anemia, together with the association of other congenital abnormalities in certain cases, suggests that the anemia is due to a fundamental inability of the marrow to produce blood cells. This is further suggested by our observation of 2 cases of this disorder, in both of which a strong familial history of similar illness was present. The excellent response of 1 of the patients to splenectomy when 4 siblings had previously died of the same disorder suggests that this operation should be seriously considered in the treatment of cases which have required numerous transfusions to maintain life. As far as we know, these are the first familial cases described in which the only hereditary anomaly was the hypoplasia of the marrow with resultant chronic anemia.

REPORT OF CASES

FAMILY 1 (table 1).—J. R. A., a 6 year old white boy, was first seen in consultation on Nov. 7, 1939. The boy had been delivered at full term after an uneventful pregnancy, and was the mother's sixth child. He was bottle fed for two years after birth. Although he was never perfectly well, no specific illnesses occurred until the age of 4, when his presenting symptoms first appeared.

Family History.—The mother and father, who were both of French Canadian descent were born in Nova Scotia but had lived in Massachusetts most of their lives. There were 6 pregnancies, as follows: ((1) a girl (E. M.), born in 1916, who died at the age of 10 years of "anemia"; (2) a boy (R.), born in 1918, who died at the age of 1 year of whooping cough and diarrhea; (3) a girl (G.), born in 1920, who is alive and well; (4) nonidentical twins, born in 1922—(*a*) a girl (M.), who is alive and well, and (*b*) a girl (E.), who died at the age of 10 years of "anemia"; (5) a girl (C.), born in 1925, who is alive and well, and (6) a boy (J. R.), born in 1933, the patient. There was no family history of tuberculosis, diabetes, cancer, syphilis or diseases of the blood, the liver, the spleen or the bones. The mother and the mother's brother had both had numerous epistaxes. The two siblings with "anemia" presented the following histories:

Sibling E. M. A. (pregnancy 1).—E. M. A., a girl, was born in 1916. She was well until August 1923, when she lost a large amount of blood during a severe spontaneous epistaxis. After this episode the patient became extremely pale and weak and began to lose weight. In January 1924 she was admitted to the Massachusetts General Hospital [26] for study. The laboratory data were as follows: red blood cells, 872,000 per cubic millimeter; hemoglobin, 10 per cent; white blood cells, 3,240, with 60 per cent polymorphonuclear forms (1,950 per cubic millimeter); and platelets, "much reduced." The diagnosis of a "congenitally defective bone marrow" was made. Epistaxis continued unabated despite symptomatic local therapy and transfusions. The patient was discharged home and died shortly thereafter.

Sibling E. A. (pregnancy 4b).—E. A., a girl, was born in 1922. Details about this child were not available. She, too, had severe epistaxis and pronounced pallor; she died just before the age of 10 years after repeated nasal hemorrhages.

The Present Patient, J. R. A. (pregnancy 6).—J. R. A., a boy, was born in 1933. His first symptom was epistaxis, which first occurred when he was 4 years of age, and recurred in severer form at the age of 5½ years. From this

26. The Massachusetts General Hospital kindly supplied the data on this child.

TABLE 1.—*Clinical and Hematologic Data in Family 1 (J. R. A.)*

Sibling	Symptoms	Signs	Date of Observation	Red Blood Cells, Millions	Hemoglobin, Percentage	White Blood Cells/ Cu. Mm.	P.M.N., Percentage	Platelets/ Cu. Mm.	Comment
E. M. A. born 1916	Epistaxis, pallor, weakness	Pallor	1924	0.872	10	3,240	60	Reduced	Died at 8 years
E. A. born 1922	Epistaxis, pallor	Pallor	....	.....	..	.....	..	.....	Died at 10 years
J. R. A. born 1933	Epistaxis, pallor, easy bruising	Pallor, ecchymoses, petechiae	Aug. 1939	1.67	25	1,750	51	Reduced	
			Nov. 1939	1.98	43	3,400	13	45,500	
			Feb. 1940	2.45	51	2,100	38	34,500	
			July 1940	1.70	45	3,100	18	.....	
			Oct. 1940	1.86	28	2,900	48	.....	
			Dec. 1940	2.25	51	2,700	..	22,500	
			Jan. 1941	1.60	39	3,600	..	40,000	Died at 8 years

time on, epistaxes were frequent and lasted from thirty minutes to one and a half hours. A tendency to easy bruising was also present. In August 1939, at the age of 6 years, the patient was admitted to another hospital because of severe epistaxis which had lasted for six hours. Physical examination revealed a pallid, poorly developed child. There was a soft systolic murmur over the entire precordium. The liver, spleen and lymph nodes were not palpable. Numerous ecchymoses and petechiae were present over the entire body. Roentgenograms of the abdomen, the chest and the long bones revealed no abnormalities. Laboratory data included the following: red blood cells, 1,670,000; hemoglobin, 25 per cent; white blood cells, 1,750, with 51 per cent polymorphonuclear cells; and platelets, "scarce." A bone marrow puncture was considered normal. The epistaxis ceased with local pressure, only to recur intermittently in the following months.

On November 7 the boy was referred for investigation. Physical examination revealed malnutrition, tiny cervical and small axillary nodes on the left, a precordial systolic murmur and no hepatosplenomegaly. There were no other physical abnormalities except for an undescended right testicle.

Laboratory data (table 1) were as follows: red blood cells, 1,980,000; hemoglobin, 43 per cent; white blood cells, 3,400, with 13 per cent polymorphonuclear forms, 71 per cent lymphocytes and 13 per cent monocytes; and platelets, 45,500 per cubic millimeter (normal 500,000). The serum bilirubin was 0.7 mg. per hundred cubic centimeters. Reticulocytes numbered 2.4 per cent.

Sternal marrow puncture revealed markedly hypoplastic marrow with a decreased number of megakaryocytes. Granulopoiesis and erythropoiesis were orderly and qualitatively normal, but quantitatively markedly diminished. A differential count of the cells was as follows:

	Percentage
Myeloblasts	0.4
Myelocytes	15.0
Metamyelocytes	6.8
Band forms	4.0
Polymorphonuclear forms	17.2
Lymphocytes	12.4
Plasma cells	0.2
Erythrogones	1.6
Basophilic normoblasts	9.6
Polychromatophilic normoblasts	12.8
Orthochromatic normoblasts	20.0

Ratio of granulocytes to red cells: 1 to 1.0 (normal 2.5 to 1)

For several months the patient was treated with various combinations of liver extract injections, yeast and iron medications, without effect. He was then hospitalized at the Boston Floating Hospital for various therapeutic maneuvers.

First Admission (February 1940): Physical examination revealed no change from November 1939. Laboratory data included: red blood cells, 2,450,000; hemoglobin, 51 per cent; white blood cells, 2,100, with 38 per cent polymorphonuclear cells, and platelets, 34,500 per cubic millimeter. The reticulocytes varied from 3.4 to 6.8 per cent. The average excretion of fecal urobilinogen was 73 Gm. per day, giving a hemolytic index of 17.2 (normal).

An experimental course of therapy with injections of "yellow bone marrow concentrate" (Armour) was undertaken, and the patient received 1 cc. of the material intramuscularly every day for a period of sixteen days. There was no change in any of the blood values with the patient on this regimen. He was discharged in three weeks unchanged.

Second Admission (July): The patient was readmitted because of continued weakness and pallor. Physical examination revealed no change. The red cell count was 1,700,000, the hemoglobin 45 per cent and the white cell count 3,100, with 18 per cent polymorphonuclear forms. Two transfusions of whole blood were given with but slight effect on the hematologic values.

Third Admission (October): The patient had had two infections of the upper respiratory tract since discharge, but no hemorrhagic episodes. Physical examination showed no change. Laboratory data were as follows: red blood cells 1,860,000, hemoglobin 28 per cent, and white blood cells 2,900, with 48 per cent polymorphonuclear cells. Four blood transfusions had but little effect on the blood values.

Fourth Admission (December): There were no new symptoms or physical findings. Laboratory data included a red cell count of 2,250,000, a hemoglobin value of 51 per cent, a white cell count of 2,700 and a platelet count of 22,500. A bone marrow puncture showed even more hypoplasia than in November 1939. Megakaryocytes were virtually absent. There were several centers of erythropoiesis and granulocytopoiesis but their number was markedly diminished. The differential count of the marrow cells was as follows:

	Percentage
Myeloblasts	0.4
Myelocytes	17.4
Metamyelocytes	3.0
Band forms	3.0
Polymorphonuclear cells	13.0
Lymphocytes	19.0
Plasma cells	1.0
Erythrogones	1.4
Basophilic normoblasts	8.0
Polychromatophilic normoblasts	14.0
Orthochromatic normoblasts	19.8
Ratio of granulocytes to erythroblasts: 1 to 1.2	

It was decided to determine the effect of transfusion of bone marrow, a procedure which had recently been recommended.[27] A compatible donor with polycythemia vera was selected because of extraordinarily high megakaryocyte and platelet levels. The donor also had moderate elevations in the erythrocyte and leukocyte counts. With the donor and the recipient under procaine hydrochloride anesthesia, sternal punctures were performed on both, our regular technic and the simple sternal puncture needle being used. Four cubic centimeters of material were aspirated into a syringe from the donor's marrow and immediately injected into the patient's marrow cavity. The procedure was done on two occasions two days apart. The hematologic counts before and after these maneuvers were as follows:

	R.B.C.	Hgb.	W.B.C.	P.M.N.	Platelets	Reticulocytes
December 18	2,250,000	51%	2,700	1,020	22,500	4.1%
December 19 First marrow transfusion				(38%)		
December 21. Second marrow transfusion						
December 23	2,240,000	49%	4,000	1,180 (27%)	39,000	5.0%

27. Morrison, M., and Samwick, A. A.: Intramedullary (Sternal) Transfusion of Human Bone-Marrow, J. A. M. A. **115**:1708 (Nov. 16) 1940.

There was thus no essential change in any of the blood values as a result of these marrow transfusions.

Fifth (Final) Admission (January 1941): There were two spontaneous epistaxes in the first week of January 1941. Physical examination revealed no change. Laboratory data included a red cell count of 1,600,000, a hemoglobin value of 39 per cent, a white cell count of 3,600 and a platelet count of 46,000. The boy was discharged after symptomatic therapy, essentially unchanged.

At home the patient was confined to bed for several weeks. He became emaciated, complained of constant fatigue and weakness and had repeated and more frequent epistaxis. In June he became comatose and died within two days. Autopsy was not performed.

Comment.—In this family of 6 siblings who lived beyond the first year of life, 3 had similar disorders characterized by pallor, hemorrhagic diathesis and death before the age of 10 years. Hematologic examination in 2 of these children showed pancytopenia (reduction in red cells, neutrophils and platelets), and in both cases the pancytopenia was attributable to hypoplasia of the marrow. In the case of J. R. A., therapy with various antianemic substances including iron, liver, yeast, blood and bone marrow was without effect. It is of interest that the mother made the diagnosis of fatal anemia with the appearance of the first epistaxis at the age of 4 years and had the constant fatalistic idea that death would occur before the age of 10 years.

FAMILY 2 (table 2).—R. A. G., a 10 year old white boy, was admitted to the Boston Floating Hospital on Oct. 9, 1944 because of bleeding from the gums and the nose. The patient was born normally after an uneventful pregnancy. At the age of 1 year he had frequent crying spells during which he seemed to be in great pain. These symptoms disappeared in a few weeks.

Family History.—The mother and father, who were both of French Canadian descent, had lived in Maine all their lives. There were 17 pregnancies. Three had ended in spontaneous abortions at the third month; 4 children had died before the age of 10 years of "anemia"; 9 children were alive and well; and 1 child was the patient. There was no history in the mother's or the father's family of disease of the blood, spleen, liver or bones. The 4 siblings with anemia and the present patient presented the following histories:

Sibling R. G.—R. G., a boy, was born in 1924. He was always considered languid and pale from infancy. At the age of 6 years, the listlessness and pallor became pronounced, and the boy began to bleed freely from the nose several times a year. These epistaxes were difficult to control. Easy bruising was noted from the age of 6. In February 1932, at the age of 8, the child was admitted to the Rumford Community Hospital, Rumford, Maine,[28] where examination revealed extreme pallor but no other physical abnormalities. Laboratory data were as follows (table 2): red blood cells, 1,270,000; hemoglobin, 23 per cent; white blood cells, 3,200, with "relative lymphocytosis," and platelets, 110,000 (normal 250,000). There was little response to several transfusions.

No essential change occurred from February to August. At this time the red cell count was 770,000, the white cell count was 2,000 and there were few platelets in smears. Treatment with iron, sodium cacodylate and a high protein diet was completely ineffective. The patient died in September of pneumonia. Autopsy was not performed.

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28. The Rumford Community Hospital, Maine, kindly supplied the data on this and the following 3 siblings.

TABLE 2.—*Clinical and Hematologic Data in Family 2 (R. A. G.)*

Sibling	Symptoms	Signs	Date of Observation	Red Blood Cells, Millions	Hemoglobin, Percentage	White Blood Cells/Cu. Mm.	P.M.N., Percentage	Platelets/Cu. Mm.	Comment
R. G. born 1924	Languor, pallor, bruising, epistaxes	Pallor	1932	1.27	23	3,200	..	110,000	
				2.0	47	2,000	..	130,000	
				0.77	..	2,000	..	.....	Died at 8½ years
E. G. born 1927	Epistaxes, pallor, hematemesis, bloody diarrhea, bleeding gums	Pallor, emaciation	1934	1.07	25	2,300	42	Rare	
			1934	1.01	18	900	..	.....	
			1936	0.48 to 2.60	10 to 50	2,900 to 3,900	18 to 50	20,000	Died at 9 years
C. G. born 1929	Pallor, epistaxis, bleeding gums, weight loss	Pallor, emaciation	1936	1.79	45	2,900	..	.....	
			1937	0.87	20	4,000	21	.....	
				1.52	30	2,100	33	.....	Died at 8 years
P. G. born 1932	Epistaxes	Pallor	1936	2.7	60	3,750	..	.....	
			1938	1.6	35	2,500	29	.....	
			1939	3.6	70	.....	..	.....	
				1.03	35	1,400	84	.....	
				4.4	75	2,900	18	.....	After 900 cc. blood
				1.67	35	1,750	..	.....	Died at 8 years
R. A. G. born 1934	Epistaxes	Pallor	1944	1.21 to 2.28	28 to 65	1,700 to 3,400	32 to 58	1,600 to 4,140	Presplenectomy
			1944	2.66 to 3.16	55 to 63	2,750 to 4,100	30 to 50	33,000 to 183,000	Postsplenectomy
			July 1945	2.45	63	2,850	22	113,000	

Sibling E. G.—E. G., a girl, born in 1927, was pale from birth. She had no illnesses, however, except for whooping cough, until the age of 7 years, when she was taken from kindergarten because of weakness and pallor. On Feb. 10, 1934 she began to bleed spontaneously from the left nostril. The bleeding continued despite packing, and the child was hospitalized a few hours later. Examination showed no physical abnormalities except the bleeding nose and the generalized pallor. The liver, the spleen and the lymph nodes were not enlarged, and no petechiae or ecchymoses were present. Laboratory data were as follows (table 2): red blood cells, 1,070,000; hemoglobin, 25 per cent; white blood cells, 2,330, with 42 per cent polymorphonuclear cells; and platelets, greatly diminished on smears. The bleeding stopped shortly after hospitalization. Local therapy consisted of packing and the use of hemostatic serum. Iron by mouth and calcium resulted in no change in the blood counts.

From 1934 to 1935 the child did fairly well at home. Only one slight epistaxis occurred during this time. She made an excellent recovery from rubella and measles. In December 1935 she again began to have nosebleeds. These were rather severe and lasted as long as one and a half hours. Oozing of blood from the mouth also occurred. In January 1936 she was rehospitalized. Except for pallor and some crusted blood at the nose, physical examination again revealed no abnormality. The red cell count was 480,000 per cubic millimeter, the hemoglobin 10 per cent, the white cell count 3,900, with 50 per cent polymorphonuclear cells, and the platelet count 20,000. There was moderate response to two blood transfusions totaling 395 cc. of blood but none to liver extract, iron or injections of whole blood intramuscularly. At the time of discharge, the red cell count was 2,390,000 and the hemoglobin was 45 per cent.

The child remained essentially unchanged from March to September 1936. In October she died of pneumonia. There was no autopsy.

Sibling C. G.—C. G., a girl, was born in 1929. At the age of 3 years the child had "rheumatic fever" and from that time on was always pale. Whooping cough occurred at the age of 5½ and measles at the age of 6. In February 1936 a routine blood cell count was as follows: red blood cells, 1,790,000; hemoglobin, 45 per cent, and white blood cells, 2,900. At the age of 7½ (1937) she had a spontaneous nasal hemorrhage which continued for three hours. Three months later the gum around a loose tooth bled for almost five hours. In April 1937, at the age of 8 years, she was admitted to the Rumford Community Hospital. At this time the red cell count was 870,000, the hemoglobin 20 per cent, the white cell count 4,000, with 21 per cent neutrophils. Physical examination showed pallor. The liver, spleen and lymph nodes were not palpable. The child died in July of pneumonia. Autopsy was not performed.

Sibling P. G.—P. G., a girl, born in 1932, had pneumonia at 11 months, and whooping cough, measles and rubella before the age of 5 years. During investigation of the entire family in 1936 she was found to have the following counts: red blood cells, 2,700,000; hemoglobin, 60 per cent, and white blood cells, 3,750. In October 1937, at the age of 5½, she had her first spontaneous nosebleed. In March 1938 epistaxis recurred and required hospitalization. Physical examination revealed blood clots in both nares and pronounced pallor. The liver, spleen and lymph nodes were not enlarged. The blood counts were as follows: red blood cells, 1,600,000; hemoglobin, 35 per cent; white blood cells, 2,500, with 29 per cent polymorphonuclear cells. There was slight response to a transfusion of 400 cc. of whole blood. The patient's condition remained unchanged from April 1938 to January 1939, when she was readmitted for transfusion. At this time the red cell count was 1,030,000, the hemoglobin 25 per cent and the white

cell count 1,400, with 84 per cent polymorphonuclear cells. Two transfusions totaling 900 cc. of whole blood were given with some effect. From January to September 1939 the child had several spontaneous epistaxes. The blood counts remained essentially unchanged. The patient died at home of pneumonia in July 1940. There was no autopsy.

The Present Patient, R. A. G.—R. A. G., a boy, was born in 1934. His first symptom was epistaxis, which first occurred at the age of 3 years in mild form. At the same time a tendency to easy bruising was noted. There were no further symptoms until two months before hospitalization (age 10 years), when the boy began to bleed suddenly from the gums; he continued to do so intermittently up to the time of admission. Three weeks before admission epistaxis occurred and continued for three hours. A transfusion of 500 cc. of whole blood was given at this time.

Physical examination revealed a pale, thin underdeveloped boy in no acute or chronic distress. He seemed depressed and inhibited. There was mild conjunctival inflammation. The scleras were white. The ears and nose were normal. The oral mucosa was pale. The tongue was slightly smooth and pale. Numerous submucosal hemorrhages were present in the lower gingival areas, and blood oozed constantly from the mouth. The lungs and heart showed no abnormalities. The liver and spleen were not palpable; there were no abdominal abnormalities. The left testicle was not palpated in the scrotum or the inguinal canal; the right was at the external ring and could be palpated into the scrotum. No lymph nodes were palpable in the cervical, axillary or inguinal regions. No petechiae were present, but old ecchymoses were seen over the dorsum of the left hand and over the right thigh.

Laboratory data were as follows: red blood cells, 1,380,000; hemoglobin, 4 Gm. (29 per cent); white blood cells, 1,800, with 65 per cent polymorphonuclear neutrophils; and platelets, 4,140 (normal 500,000). Reticulocytes numbered 8.6 to 10.7 per cent. Serum bilirubin was 0.9 mg. per hundred cubic centimeters. The bleeding time (Duke method) exceeded twenty-three minutes (normal under five minutes). The coagulation time (Lee-White method) was five minutes (normal). Clot retraction was absent. The blood sedimentation rate (Westergren method) was 85 mm. per hour (normal 20 mm.). The hypotonic fragility test showed beginning hemolysis at 0.44 per cent and complete hemolysis at 0.24 per cent saline solution (normal).

An examination was made of the bone marrow, first by the puncture-aspiration technic and then by the trephine technic. The findings at both were identical. The marrow was slightly hypocellular, with normal development of granulocytes, relative normoblastic hyperplasia and normal numbers of megakaryocytes which were, in general, smaller than normal and showed virtually no formation of platelets. A differential count of the marrow was as follows:

	Percentage
Myeloblasts	0.4
Myelocytes	20.0
Metamyelocytes	6.0
Band forms	6.0
Polymorphonuclear cells	12.0
Lymphocytes	0.8
Plasma cells	1.2
Erythrogones	3.2
Basophilic normoblasts	21.0
Polychromatophilic normoblasts	14.6
Orthochromatic normoblasts	14.8

Ratio of granulocytes to erythroblasts: 1 to 1.3 (normal 2.5 to 1)

Roentgenologic examination of the chest, abdomen and long bones revealed no abnormalities.

The patient was given four transfusions of whole blood, totaling 625 cc. with no essential change in the blood picture. Because of the striking family history with death of 4 siblings given conservative treatment, the presence of reticulocytosis, indicating some degree of marrow activity, and the presence of a fair number of megakaryocytes, showing diminished formation of platelets (thus resembling somewhat the findings in idiopathic thrombopenic purpura), the possibility that splenectomy might be of therapeutic benefit suggested itself. The spleen was removed on Nov. 1, 1944, transfusions of whole blood (totaling 1,035 cc.) being given both before and after the operation. The spleen was not enlarged and pathologically showed no abnormalities. No marked cessation of bleeding occurred when the splenic pedicle was ligated.

Postoperatively, the bleeding from the gums and the oral mucosa ceased completely. The patient did well. The platelet level rose from 4,000 before operation to 183,000 on November 17 and then became stabilized at about 97,000 (December 9). The red cell count rose from 1,860,000 and 2,280,000 before the operation to a peak of 3,160,000 on Nov. 17, 1945 and then became stabilized at about 3,000,000 on December 6. There was a parallel rise in hemoglobin concentration. The reticulocyte count fell gradually from preoperative levels of 5 to 10 per cent to a value at discharge of 1.9 per cent. The leukocyte count rose from a preoperative value of about 2,000 per cubic millimeter to a postoperative average of about 3,900 (December 6) with about 50 per cent neutrophils.

There was a great improvement in the patient's psychic state. Prior to the operation he seemed surly, uncommunicative and depressed. With cessation of the bleeding, he began to talk freely, smiled often, took an interest in his surroundings, ate better and began to gain weight. Administration of vitamin B complex, iron (elixir "feosol," 1 drachm [4 cc.] three times a day) and 2 ounces (60 cc.) of liquid extract of liver with iron (Valentin Meat Juice Company) three times a day continued. He also received 15 units of concentrated liver extract by injection twice a week.

The boy was discharged home on Dec. 9, 1944 greatly improved. On July 13, 1945, eight months after splenectomy, the patient returned for reexamination. He had remained in good health and had shown no bleeding whatever since discharge from the hospital. The mouth and the nose were in good condition. The liver was not felt. The splenectomy scar was well healed. No lymph nodes were palpable anywhere. There were no petechiae or ecchymoses. Examination of the blood at this time gave the following results: red blood cells, 2,450,000; hemoglobin, 9.8 Gm. (63 per cent); white blood cells, 2,850, with 22 per cent neutrophils, and platelets, 113,000 per cubic millimeter (normal 500,000). Reticulocytes numbered 3.5 per cent. The bleeding time was five minutes. The coagulation time was five, nine and thirteen minutes (normal). Clot retraction was normal. A sternal puncture showed moderate hypoplasia with quantitatively diminished granulopoiesis, erythropoiesis and thrombopoiesis. Qualitatively, development of all these cells was normal. No pathologic cells appeared in the preparations. A differential count of the marrow showed:

	Percentage
Myeloblasts	0.2
Myelocytes	22.0
Metamyelocytes	4.0
Band forms	6.0
Polymorphonuclear cells	12.0
Lymphocytes	5.0
Plasma cells	0.8
Erythrogones	2.0
Basophilic normoblasts	8.0
Polychromatophilic normoblasts	16.0
Orthochromatic normoblasts	24.0

Ratio of granulocytes to erythroblasts: 1:1.1

Comment.—In this family of 14 siblings, 5 had similar disorders, characterized by pallor, hemorrhagic diathesis, and pancytopenia. In 4 of these children, death occurred before the age of 10 years. Therapy with various antianemic substances (iron, liver, yeast and blood) was without effect. In the fifth child splenectomy was undertaken, and resulted in elimination of the patient's complaints, although there was little change in the hematologic values except for the rise in platelets. This patient is living and well and has already attained an age unreached by his 4 siblings.

COMMENT

The outstanding feature of the cases presented in this paper is the familial character of the disorder, identical symptoms, signs and blood pictures occurring respectively in 3 of 6 siblings in one family, and in 5 of 14 siblings in the other. It is of interest that both families were of French Canadian origin. The familial character of these cases and their similar nature to previously described cases of hypoplastic anemia suggest strongly that the hypoplastic anemia of childhood is a congenital developmental defect of the bone marrow. Since no developmental defects other than the bone marrow lesion were present, it may be postulated that only the genetic mechanism responsible for the development of normal marrow tissue was so disturbed that hemopoiesis was more or less permanently damaged. This might conceivably be due to the presence of a recessive characteristic in one or both parents. Certain investigations in the literature suggest that idiopathic aplastic anemia may sometimes be the culmination (in certain persons) of a familial tendency to neutropenia, anemia or thrombopenia. Thus Huber [29] examined the families of 3 patients who died of aplastic anemia and found that 6 of 23, 4 of 16 and 3 of 18 relatives respectively showed a tendency to neutropenia or anemia, whereas 11 relatives of a patient with aplastic anemia due to trichloroethylene showed no such abnormalities.

The familial nature of this disorder links it, perhaps, to the cases of familial anemia described by Fanconi.[19] In Fanconi's syndrome, however, marrow hypoplasia was merely one of multiple congenital defects. Apparently, in these cases multiple genetic mechanisms were simultaneously disturbed, so that developmental aberrations occurred in the skeletal system (microcephaly, absence of thumbs), the nervous system (exaggerated deep tendon reflexes), the eyes (strabismus), the urinary system (absent kidney) and the genitalia (testicular hypoplasia), as well as in the hemopoietic system (hypoplasia of the marrow). Most instances of Fanconi's syndrome show familial incidence,[30] although sporadic cases have also been reported.[31] The familial nature of the cases of hypoplastic anemia reported in the present paper is outstanding,

29. Huber, H.: Stammbaumuntersuchungen bei Panmyelophthisekranken, Klin. Wchnschr. **18**:1145, 1939.

30. Fanconi.[19] Emile-Weil.[20] Hjorth.[21] Dacie and Gilpin.[22]

31. Emile-Weil.[20] Uehlinger.[23] van Leeuwen.[24] Estren and Dameshek.[25]

but there is nothing to suggest the presence of any developmental anomalies outside of the bone marrow.[32] These cases may therefore be considered as the first authenticated instances in which marrow hypoplasia occurred as a familial characteristic without other developmental abnormalities.

All cases in our two families showed pancytopenia, i. e., a simultaneous reduction in red cells, leukocytes, granulocytes and platelets. From the standpoint of differential diagnosis this complex may be due to various conditions, as follows: (1) a disturbance of the bone marrow —hypoplasia, aplasia or replacement by abnormal cells; (2) a deficiency syndrome, in which the marrow is "full" but there is a deficiency of hemopoietic substance which interferes with normal production of blood cells; (3) "hypersplenism," in which the spleen exerts an unusual degree of inhibitory effect on the normal hemopoietic processes of the marrow. In almost all instances of hypersplenism the spleen is definitely enlarged.

The cases here considered showed no evidence either clinically or at examination of the bone marrow of replacement of the marrow by leukemic or sarcomatous cells. There was no evidence of a deficiency syndrome. Since the spleen was in no case enlarged, hypersplenism seemed unlikely. This left as the only likely possibility the presence of a hypoplasia of the marrow. Examination of the marrow (sternal puncture) in patient J. R. A. showed generalized quantitative hypoplasia of all elements, although qualitatively development was orderly. In patient R. A. G., there was also evidence of hypoplasia of the marrow with a reduction in all the blood-forming elements.

Despite these findings, however, a certain degree of reticulocytosis was present in both patients—3.4 to 6.8 per cent in patient J. R. A. and 8.6 to 10.7 per cent in patient R. A. G. There was no indication of hemolysis to which the reticulocytosis might be secondary. These findings indicated, therefore, that some degree of regenerative activity was present either in some sections of the marrow or in the spleen (extramedullary hemopoiesis or myeloid metaplasia). Since splenomegaly was lacking and no early white cells were found in the blood, myeloid metaplasia could almost certainly be ruled out. It was concluded that the fundamental difficulty was inability of the marrow to function at a normal level but that some activity of the marrow was present, and that a certain amount of splenic inhibition might be superimposed on the congenital defect. The gamut of therapy was utilized before removal of this postulated inhibitory factor was undertaken in patient R. A. G.

All sorts of medications and maneuvers have been utilized in the treatment of hypoplastic anemia of childhood. Absence of therapy has occasionally been followed by a spontaneous and sustained remission.[18]

32. In both patient S. R. A. and patient R. A. G. the testes were partially undescended but not apparently hypoplastic.

This occurrence in true marrow aplasia is rare; it appears to be more common in the temporary pancytopenias which frequently accompany acute infections. In the absence of therapy, however, death is the rule. Periodic blood transfusions are commonly required and have been the usual form of treatment. As a rule, they were given every four to six weeks,[13] depending on the rapidity of recurrence of symptoms and severe anemia. In cases in which transfusions were the only mode of treatment, death followed their cessation.[10] Antianemia substances of all types (iron, liver, arsenic, etc.) have been useless in the therapy of this disorder. Certainly, little if any value could be expected from their use in a condition in which the marrow is basically unable to function at a sufficiently high level. Epinephrine was utilized with apparent effectiveness by Gibson.[33] This author reported an 11 year old girl with idiopathic aplastic anemia who required seven blood transfusions in nineteen months. At this time, she was given daily injections of epinephrine, with pronounced alleviation of symptoms and improvement in blood counts. Although her hematologic values never returned to normal, she was maintained satisfactorily on daily injections of epinephrine for six years, when she died of hemorrhagic chickenpox.

"Yellow bone marrow concentrate" was given to 1 of our patients with no effect subjectively or objectively. Bone marrow transfusions were first recommended by Morrison and Samwick,[27] who reported encouraging results in 1 case of aplastic anemia. González-Guzmán [34] and Yllades [35] continued the technic and elaborated on it. The latter author found good results in various macrocytic and hypochromic anemias, but mentioned poor results in pancytopenias. Vaughan [36] and more recently Bernard [37] found no results in cases of aplastic anemia. The same was true of marrow given intravenously by Osgood.[38] One of our patients (J. R. A.) received large amounts of polycythemic marrow (i. e. marrow which was the direct antithesis of the patient's own marrow) with no improvement.

Splenectomy has been performed in an occasional case of aplastic anemia, both in adults and in children. The operation was followed by death in ten hours in Illing's case in a child.[14] It had no effect in Rosen-

33. Gibson, A. G.: The Action of Adrenalin in Aplastic Anemia, Lancet. **2**: 948, 1926; Aplastic Anemia: Sequel to a Case Report, ibid. **2**:181, 1931.

34. González-Guzmán, I., cited by Yllades.[35]

35. Yllades, L. S.: Ultimos estudios sobre la transfusión de médulla, Medicina, México **23**:75, 1943.

36. Vaughan, S. L.: Aplastic Anemia, New York State J. Med. **42**:978, 1942.

37. Bernard, J.: Les injections intra-médullaires chez l'homme, Sang **17**:61, 1945.

38. Osgood, E. E.; Riddle, M. C., and Mathews, T. J.: Aplastic Anemia Treated with Daily Transfusions and Intravenous Marrow, Ann. Int. Med. **13**: 357, 1939.

thal's 3 adults [39] or in van Leeuwen's child with Fanconi's syndrome.[24] There was a transient beneficial effect in Gottlieb's patient,[40] aged 19, but relapse and death followed after a brief and incomplete remission.[41] Whitby and Britton [42] mentioned 6 cases of aplastic anemia in which splenectomy was beneficial, but did not give details. Dacie and Gilpin [22] performed splenectomy in 1 of their cases of familial hypoplastic anemia of the Fanconi type, and reported definite and sustained improvement, so that transfusions which had to be given regularly before splenectomy were no longer required after the operation.

The presence in patient R. A. G. (family 2) of a certain degree of reticulocytosis, indicating that marrow activity was not completely lacking, together with the presence of a number of megakaryocytes showing lack of platelet formation and therefore resembling more or less the findings in idiopathic thrombocytopenia purpura, suggested the possibility that splenectomy might be of therapeutic value. This procedure was therefore decided on, especially in view of the fact that death had occurred in 4 siblings afflicted with the same disorder. The therapeutic result, although it failed to produce a complete restitution to normal blood values, was sufficiently marked to cause an almost immediate cessation in the hemorrhagic tendency together with definite increases in red cells, leukocytes and platelets. The result may be classed as the nonspecific effect of splenectomy, similar to that seen in normal persons in whom striking and sustained increases in platelets with moderate rises in leukocytes and granulocytes occur following splenectomy.[43] Removal of the normal spleen thus appears to remove a factor which exerts a normal degree of regulatory (inhibitory) effect on red cells, granulocytes and platelets at the marrow.

In cases of complete or almost complete aplasia of the marrow and in cases in which a reduction of blood platelets is accompanied by a reduction or absence of megakaryocytes in the marrow, it would not be expected that splenectomy would be of value. However, when megakaryocytes are present to some degree in the marrow, and particularly when lack of platelet production in the megakaryocytes can be demonstrated, and when, in addition, the red cell elements show some evidence of regenerative activity, splenectomy may be seriously considered as offering a possibly beneficial therapeutic result.

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39. Rosenthal, N.: Hematological Aspects of Agranulocytosis and Other Diseases Accompanied by Extreme Leukopenia, Am. J. Clin. Path. **1**:7, 1931.

40. Gottlieb, R.: Myeloid Insufficiency, Ann. Int. Med. **7**:895, 1934.

41. No marrow studies or autopsy were performed in this case and the exact nature of the pancytopenia therefore remains obscure.

42. Whitby, L. E. H., and Britton, C. J. C.: Disorders of the Blood, ed. 5, Philadelphia, The Blakiston Company, 1946, p. 384.

43. Singer, K.; Miller, E. B., and Dameshek, W.: Hematologic Changes Following Splenectomy in Man, Am. J. M. Sc. **202**:171, 1941.

It is of interest in this regard that, although hypoplasia of the marrow was present in these patients, both showed high reticulocyte counts. The occurrence of reticulocytosis in true aplastic or hypoplastic pancytopenias is not commonly recognized, but its presence, especially in children, is definite. In such cases, removal of the spleen seems to eliminate the inhibitory (regulatory) mechanism which the spleen normally exerts on the marrow. The result is an increase in the liberation of erythrocytes and granulocytes from the marrow, and especially an increase in the production of platelets within the megakaryocytes and their delivery into the blood stream. In normal persons, these changes produce no particular effect. In these patients, however, there may be sufficient rise in the count of red cells, neutrophils or platelets after splenectomy that the symptoms of anemia, neutropenia or thrombopenia may be alleviated. The effect is especially to be desired in cases in which the main complaints result from thrombopenia (patient R. A. G.), for the mild thrombocytosis occurring after splenectomy may be sufficient to prevent further bleeding. This was the actual result obtained in this case.

In many cases of hypoplastic anemia of childhood, the use of periodic transfusions is usually sufficient to maintain the child in relatively good clinical and hematologic condition. In others, the marrow is evidently so hypocellular that transfusions have but little effect and the course is a consistently downhill one. In a few cases, which are evidently intermediate between these extremes of mildness and severity, splenectomy may be of distinct value by eliminating a factor which is normally inhibitory to the marrow.

SUMMARY

Two families with familial hypoplastic anemia are presented. Three of 7 siblings in the first family and 5 of 14 siblings in the second family had apparently identical disorders characterized by peripheral pancytopenia, pallor, weakness and a bleeding tendency. Hypoplasia of the marrow was present in those cases in which the marrow was examined.

These are the first recorded instances in which hypoplasia of the marrow has been noted as the sole familial trait. The closest similar cases in the literature are those of Fanconi, in which hypoplasia of the marrow was one of several congenital and familial abnormalities. Familial hypoplastic anemia must be added to the other hematologic conditions in which a heredofamilial incidence occurs.

Splenectomy in 1 of the patients resulted in pronounced improvement. The rationale for splenectomy in certain cases of congenital hypoplastic anemia is discussed.

Case Reports

CARDIAC COMPLICATIONS IN SALMONELLA INFECTIONS

BERNARD HAROLD SHULMAN, M.D., BROOKLYN

THE GENUS Salmonella is a serologically related group of organisms occurring in human beings and animals. They are primarily enteric pathogens, but may produce various clinical pictures. These have been described as "typhoid-like," gastroenteritis or enterocolitis and sepsis with bacteremia, with or without localization. The latter is often secondary to the enteric type, and probably accounts for the parenteral complications that are encountered. These include meningitis,[1] osteomyelitis,[2] pneumonia [3] and focal abscesses.[4]

In infants and children, Salmonella infections usually manifest themselves as enteric infections, with diarrhea as the presenting symptom. Cases may occur sporadically or epidemically. The presence of bacteremia is of serious import and introduces the possibility of parenteral localization.

In addition to the complications already noted, the occurrence of cardiac complications is more than a mere possibility. The literature contains several reports of such cases (the table). Gouley and Israels [5] reported a case of acute bacterial endocarditis due to Salmonella suipestifer in a 4 year old white boy, with recovery. Other cases of bacterial endocarditis have been reported by Wells,[6] Meyer and Howell,[7] Read,[8] Goulder, Kingsland and Janeway,[9] Seligmann, Saphra and Wasser-

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1. Bahrenberg, J. H., and Ecker, E. E.: Meningitis Due to Organisms Belonging to Salmonella Group, J. Infect. Dis. **60**:98 (Jan.-Feb.) 1937.

2. Neal, J. R.: Paratyphoid Osteomyelitis, Am. J. Surg. **43**:594 (Feb.) 1939.

3. Bullowa, J.: Paratyphoid: Two Cases with Pneumonia, M. Clin. North America **15**:1127 (March) 1932.

4. Clifton, W. F., and Werner, M.: Infection with Salmonella Suipestifer in Childhood with Report of a Case of Bilateral Subdural Abscess Ending in Recovery, Am. J. Dis. Child. **55**:553 (March) 1938.

5. Gouley, B. A., and Israels, S. L.: Salmonella Suipestifer Bacteremia with Acute Endocarditis, Arch. Int. Med. **53**:699 (May) 1934.

6. Wells, G. H.: Acute Endocarditis Produced by Bacillis Paratyphosus B, Arch. Path. **23**:270 (Feb.) 1937.

7. Meyer, J., and Howell, K.: Acute Endocarditis Caused by Bacterium Paratyphosus B, Arch. Path. **26**:368 (July) 1938.

8. Read, C. T.: Endocarditis Caused by Salmonella Suipestifer, J. Infect. Dis. **65**:262 (Nov.) 1939.

9. Goulder, N. E.; Kingsland, M. F., and Janeway, C. A.: Salmonella Suipestifer Infections in Boston: Report of Eleven Cases with Autopsy Findings in a Case of Bacterial Endocarditis Due to This Organism and Study of Agglutination Reaction in This Infection, New England J. Med. **226**:127 (Jan. 22) 1942.

man [10] and Forster.[11] The latter reviewed 2 cases, with complete heart block and auricular fibrillation, respectively. That Salmonella infections may occur in the presence of other infections has been noted, and Keeling [12] reported a case of acute bacterial endocarditis, from which both Salmonella aertrycke and Staphylococcus aureus were recovered at autopsy. An unusual case of pericarditis, pneumonitis and pleural effusion, caused by Salmonella suipestifer, was reported by Cohen, Fink and Gray.[13]

Cardiac Complications of Salmonella Infections in the Literature

Authors	Age	Organism	Probable Primary Focus	Cardiac Complications	Outcome
Gouley and Israels [5]	4 yr.	S. suipestifer	Bacteremia	Acute endocarditis	Recovered
Wells [6]	50 yr.	S. aertrycke	Pneumonia	Acute endocarditis	Died
Meyer and Howell [7]	36 yr.	Bacterium paratyphosum B	Septicemia	Acute endocarditis	Died
Read [8]	49 yr.	S. suipestifer	Ovarian cyst with bacteremia	Acute endocarditis	Died
Goulder, Kingsland and Janeway [9]	58 yr.	S. suipestifer	Enteric	Acute endocarditis	Died
Seligmann, Saphra and Wasserman [10]	All adults	S. choleraesuis	Bacteremia	Subacute bacterial endocarditis	Died
		S. choleraesuis	?	Subacute bacterial endocarditis	Died
		S. typhimurium	?	Subacute bacterial endocarditis	Died
		S. choleraesuis	?	Acute endocarditis	Died
		S. choleraesuis	?	Acute endocarditis	Died
		S. typhimurium	?	Acute endocarditis	Died
		S. thompson	?	Acute endocarditis	Died
Forster [11]	47 yr.	S. suipestifer	Septicemia	Acute endocarditis with complete heart block	Died
Forster [11]	31 yr.	S. suipestifer	Septicemia	Acute endocarditis with auricular fibrillation	Died
Keeling [12]	37 yr. (case 2)	S. aertrycke and Staph. aureus	Bacteremia; exposed to patient with Salmonella septicemia	Acute endocarditis (both organisms recovered at autopsy)	Died
Cohen, Fink and Gray [13]	36 yr.	S. suipestifer	Bacteremia, pneumonitis and pleural effusion	Acute pericarditis	Recovered
Shulman	2 mo.	S. enteritidis	Enteric	Acute paroxysmal auricular tachycardia	Recovered

10. Seligmann, E.; Saphra, I., and Wasserman, M.: Salmonella Infections in Man, Am. J. Hyg. **38**:226 (Nov.) 1943; personal communication to the author.

11. Forster, D. E.: Fatal Bacterial Endocarditis Due to Salmonella Suipestifer, Am. J. M. Sc. **197**:234 (Feb.) 1939.

12. Keeling, J.: Salmonella Septicemia: A Report of Seven Cases, New Zealand J. Med. **41**:254 (Dec.))1941.

13. Cohen, L.; Fink, H., and Gray, I.: Salmonella Suipestifer Bacteremia with Pericarditis, Pneumonitis, and Pleural Effusion, J. A. M. A. **107**:331 (Aug. 1) 1936.

This is a report of a case of acute enteritis due to Salmonella enteritidis, in a 2 month old Negro infant, with bacteremia and cardiac complication as manifested by acute paroxysmal auricular tachycardia.

REPORT OF A CASE

W. J., a 2 month old Negro boy, was admitted to the Department of Pediatrics, Long Island College Division of Kings County Hospital, on Dec. 22, 1942, because of an infection of the upper respiratory tract of six days' duration.

The birth and neonatal history were normal, and the family background was not unusual.

For the month preceding admission to the hospital, the child had been having from three to six loose nonbloody stools a day. For the week prior to entry, the infant had been coughing. During this same period he seemed to cry with each bowel movement and he vomited on several occasions.

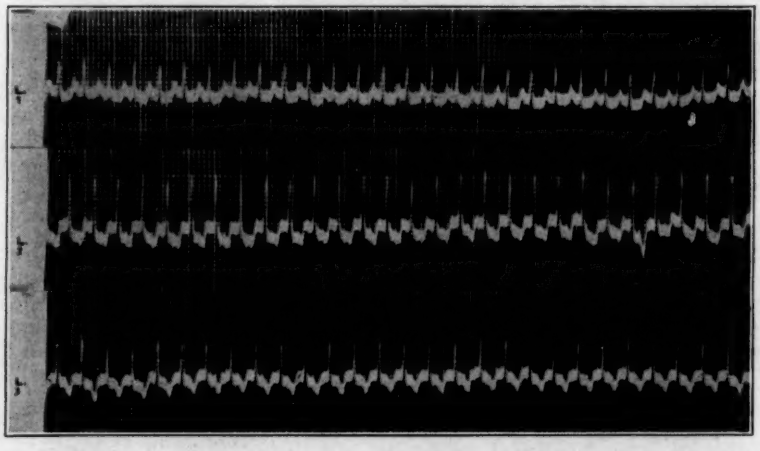

Fig. 1.—Electrocardiogram taken on the fifth hospital day, showing auricular and ventricular rates of 290 beats per minute.

On admission, the patient did not appear acutely ill. He weighed 10 pounds and 5 ounces (4,682 Gm.). The temperature was 102.0 F., and pulse and respirations were normal. The lungs were clear, and no cardiac abnormalities were observed. The only abnormalities noted were congestion of the nose and the pharynx.

The child was placed in the isolation room and started on a modified diarrhea regimen. He was placed on a lactic acid evaporated milk formula and treated for the respiratory infection. The temperature fell to 99.0 F. and remained at that level until the third day. During the first forty-eight hours, he had thirteen loose stools. One stool culture was reported positive for Salmonella enteritidis. On the third day the temperature rose to 103.5 F. A blood culture taken at this time was reported positive for Salmonella enteritidis. The blood count showed 22,320 white blood cells with 72 per cent polymorphonuclear leukocytes and 3,980,000 red blood cells with 11.5 Gm. of hemoglobin. Reexamination failed to reveal any obvious focus of infection. The cardiac findings, however, were startling. The previous rate was now replaced by a rate too rapid to count. The sounds

were ticktack in quality. There was no change in the size of the heart, and it was not possible to determine the presence of murmurs. The tachycardia was not altered by pressure on the eyeballs or the carotid sinus. An electrocardiogram was taken and revealed an auricular rate of 290 per minute, with an equal ventricular rate (fig. 1). The P-R interval was 0.06 second, and the QRS complex 0.03 second. There was no axis deviation. In lead II the S-T segment was depressed, and the T deflection negative. In lead III T was notched. A tentative diagnosis of paroxysmal tachycardia of auricular or nodal origin was made.

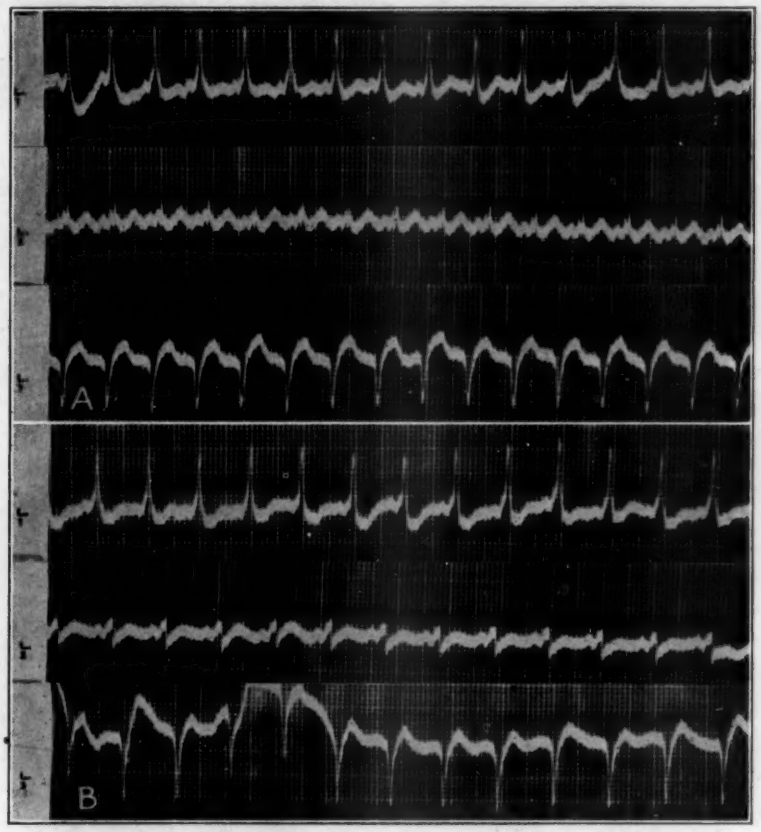

Fig. 2.—*A*, electrocardiogram taken on the seventh hospital day, showing auricular and ventricular rates of 166 beats per minute. *B*, electrocardiogram taken on the ninth hospital day, showing auricular and ventricular rates of 140 per minute.

Auricular flutter with a 1:1 block was likewise considered. A roentgenogram of the chest showed a normal cardiac contour.

The child was given an initial dose of 7½ grains (0.5 Gm.) of sulfathiazole and then given 3¾ grains (0.225 Gm.) every four hours. Chemotherapy was continued for ten days until the fifteenth hospital day, when it was discontinued. The blood level of sulfathiazole reached 5.1 mg. per hundred cubic centimeters. At the same time that chemotherapy was instituted, the child was given 1 cat

unit of digitalis intramuscularly. The pulse was taken every twenty minutes. After two hours and fifteen minutes, it slowed to a rate of 180 per minute. The temperature fell to normal. For the first time a systolic murmur could be heard at the apex. Eighteen hours after the single dose of digitalis, the pulse rate was 130 per minute and the temperature rose to 103.0 F. The murmur could still be heard. The child was given 1 cat unit of digitalis intramuscularly, and then received ½ cat unit every six hours intramuscularly. After eight such doses, the dosage was reduced to ½ cat unit daily and continued for six days. On the

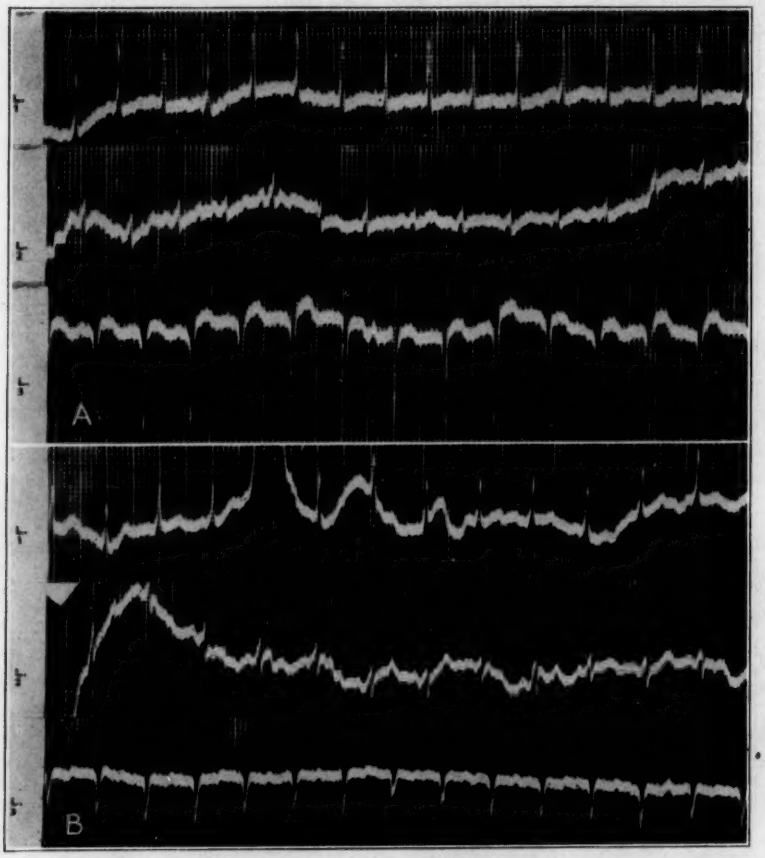

Fig. 3.—*A*, electrocardiogram taken on the fifteenth hospital day, showing auricular and ventricular rates of 160 beats per minute. *B*, electrocardiogram taken on the twenty-third hospital day, showing auricular and ventricular rates of 140 beats per minute.

eleventh hospital day, the digitalis was discontinued. The pulse at this time was 110 per minute. The murmur had disappeared.

Repeated electrocardiograms showed progressive slowing of the heart rate during this period of digitalization (fig. 1 and 2). At no time during the phase of arrhythmia did the infant show any respiratory distress or discomfort. No signs of cardiac failure appeared. The combination of digitalis, sulfathiazole and dietary control brought about a remarkable change in the patient. The temperature remained normal, and the diarrhea subsided. Repeated blood cultures were

sterile after the initial positive culture for Salmonella enteritidis. Several stool cultures were negative for the same organism.

On the thirty-eighth hospital day, the child was discharged in excellent condition. His weight on discharge was 12 pounds (5,448 Gm.). The heart rate was 120 per minute, and no murmurs could be heard.

COMMENT

Bornstein[14] stated that "the most dangerous form of Salmonella infection is the septic type, which represents about one-sixth of the sporadic cases, but which causes three-fourths of the deaths." In the 17 cases of cardiac complications in Salmonella infections listed in the table, there were 14 deaths, a mortality rate of 82.5 per cent. It is interesting to note that 2 recoveries occurred in children, including my patient. The other child who recovered was a 4 year old boy with Salmonella suipestifer bacteremia and acute endocarditis, reported by Gouley and Israels.[5]

Of the 17 cases in the English literature, there were 12 cases of acute endocarditis, 3 of subacute endocarditis, 1 of pericarditis and 1 of acute paroxysmal auricular tachycardia. Two of the patients with acute bacterial endocarditis had auricular fibrillation and complete heart block, respectively.

No single organism of the Salmonella group could be implicated in the cases reviewed, and the possibility that a cardiotoxic factor was present could not be inferred from the cases listed in the table. The etiologic agents were Salmonella suipestifer in 6 cases; Salmonella typhimurium in 2 cases; Salmonella choleraesuis in 4 cases; Salmonella thompson in 1 case; Salmonella enteritidis in 1 case, and Salmonella aertrycke in 2 cases. In one of the latter cases Staphyloccocus aureus bacteremia occurred simultaneously, and both organisms were recovered at autopsy.

SUMMARY

A case of Salmonella enteritidis enteritis in a 2 month old Negro infant is reported, with a description of the complicating bacteremia and acute paroxysmal auricular tachycardia. Serial electrocardiograms are included in the report.

The use of sulfathiazole in the treatment of the diarrhea is described. The value of the drug in this case is undetermined, though the improvement of the patient coincided with the use of the drug.

The use of digitalis to control the paroxysmal auricular tachycardia is described. Eighteen hours after the initial dose, the heart rate slowed from 290 beats per minute to 130 beats per minute. This seemed to be a therapeutic response. Maintenance doses of digitalis kept the pulse rate below 150 beats per minute, and after six days of administration of digitalis the drug was discontinued. Subsequently the pulse remained at or below 110 beats per minute.

A review of other cases of Salmonella infections with cardiac complications is included in the form of a table.

1162 Union Street.

14. Bornstein, S.: Epidemiological Aspects and Laboratory Diagnosis of Salmonella Infections, New York State J. Med. **42**:2215 (Dec. 1) 1942.

Progress in Pediatrics

INTRODUCTION TO THE PROBLEM OF PREMATURITY

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THE SUBJECT of prematurity is of great interest and concern to pediatrists. About 3,000,000 babies are currently born each year in the United States. Of these, approximately 5 per cent, or 150,000, are premature. Less than 2 per cent of the full term babies die in the neonatal period; nearly 50 per cent of the premature babies account for the same number of deaths. In absolute figures, 60,000 of the 3,000,000 full term babies and 60,000 of the 150,000 premature babies succumb

Handicaps of the Premature Infant

Physiologic Manifestations	Pathologic Exaggerations
1. Difficulties of respiration	Asphyxial attacks, aspiration pneumonia
2. Difficulties of circulation	Cyanosis of extremities, edema
3. Faulty control of body temperature	Hypothermia and hyperthermia
4. Diminished alimentary tolerance	Gastrointestinal upsets
5. Increased capillary fragility	Hemorrhage
6. Impairment of renal function	Dehydration and acidosis
7. Inadequate antenatal storage:	
Minerals and vitamins	Rickets, scurvy, anemia
Immune substances	Infections
8. Defective hemopoiesis	Anemia
9. Hepatic immaturity:	
Bilirubinemia	Jaundice
Hypoglycemia	Shock
Hypoproteinemia	Edema
Hypoprothrombinemia	Hemorrhage
10. Increased content of body water	Infections, edema
11. Retrolental fibroplasia—infants under 1,500 Gm.	Blindness
12. Incomplete development of enzyme systems	Faulty metabolism of aromatic amino acids, impaired formation of hemoglobin, reduction of carbonic anhydrase

before the first month of life. It is evident that any attempt to reduce infant mortality further must include a vigorous attack on the problem of prematurity. In New York city, 36 per cent of all infant deaths were attributable to prematurity in 1944 and 1945. Prematurity is listed as ninth in the leading causes of all deaths in the United States in 1945.

The explanation for the high mortality rate of prematurely born infants is understandable on physiologic grounds. Every newborn baby in transit from a parasitic intrauterine existence to independent life must

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Presented in a panel discussion on prematurity at the annual meeting of the American Academy of Pediatrics, Pittsburgh, Feb. 24, 1947.

make a number of adjustments to his changed environment. The success with which these adjustments are made depends on a number of factors: the health and internal state of the mother in pregnancy, the position of the fetus in utero and complications of labor and delivery requiring anesthesia and operative procedures. The most important single factor is, however, the state of maturity of the infant. Immaturity of the systems of the premature infant subjects him to a number of handicaps which are present in a far less degree in the full term infant. Some of these handicaps are listed in the table.

It is obvious that in the limited time allotted to this panel the management of only some of these handicaps can be discussed. Among the important aspects of management of premature infants are included: (1) the prevention and control of infections; (2) treatment of emergencies; (3) the control of body temperature, and (4) feeding. We are fortunate to have such competent men on the panel to discuss each of these fields.

PREVENTION AND CONTROL OF INFECTIONS

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IN THE monthly bulletin issued by the Children's Bureau in March 1942 there appeared the following statements: "Among the causes of neonatal deaths (deaths of infants under one month of age) in the report of the United Bureau of Census, premature birth played the chief role, accounting for 46 per cent of these deaths in 1940." In Chicago, except for infants born in the Michael Reese Hospital, of which the Sarah Morris Hospital for Children is a division, over 90 per cent of the 6,627 premature infants sent to the Sarah Morris Hospital for Children are brought in by a specially equipped ambulance. A total of 1,825 (27.5 per cent) died; of these deaths 55.6 per cent occurred within twenty-four hours and another 14 per cent between twenty-four and forty-eight hours after the infants were received in the station. There is a city regulation which requires a report of premature births within one hour by telephone and within twenty-four hours in writing. As a result of the early reporting and delivery of the infants to the hospital, it can be readily understood that many small and nonviable infants are received.

In appreciation of the implications of these figures, the premature birth of an infant must be viewed in the light of an emergency in order that the infant may be given its best opportunity for life and normal physical development.

In order properly to protect the infant, wherever possible premature delivery should be anticipated. If this is not possible the infant's birth should be reported immediately to the department of health so that the proper steps may be taken to protect it from the hazards which it will encounter.

It must be taken for granted that every exposure to which the premature infant is subjected tends to make it less resistant to contacts with infections, both bacterial and virus. It therefore becomes evident that the infant cannot wait until it reaches a special station before methods for its protection are inaugurated.

Personally, I am best acquainted with the activities carried out for the protection of premature infants in Chicago and the state of Illinois, of which I shall speak later. Programs with the same objectives have

Presented in a panel discussion on prematurity at the annual meeting of the American Academy of Pediatrics, Pittsburgh, Feb. 24, 1947.

been inaugurated in other cities and states. Massachusetts was the first state to provide a well organized statewide program.

The station for premature infants at the Sarah Morris Hospital for Children, established in 1922, was the first of its kind in Chicago that was willing to receive premature infants born in other hospitals and in homes. The demand for such a station is evidenced by the gradual increase in the number of premature infants admitted to the hospital from 19 in the first year of operation to 462 in 1946. During this period a total of 6,627 infants were admitted. As early as 1932 the station became overtaxed, and provision was made for the enlarging of the small station at the Cook County Hospital to provide for patients born in that hospital and for patients received from outside sources.

During 1932, the first year, the Cook County Hospital station for premature infants admitted 138 and in the year 1946 it admitted 517.

Appreciating the need for better provisions for the care of the premature infant, the Board of Health of Chicago in 1935 started the Chicago citywide plan for their care. The program inaugurated was an attempt to expand the procedures in effect at Sarah Morris Hospital for Children to meet the demands of a large metropolitan community.

For a better understanding of the causes of death of premature infants, a plan for the investigation of the cause of death of every infant in Chicago was instituted in January 1935.

It immediately became apparent that in attacking the problem of prematurity, just as in an epidemic the number of cases of contagion must be known, it would be essential to have early reports of premature births. Therefore on March 5, 1935 the Board of Health passed a regulation making premature births immediately reportable by telephone, after which there was to be written confirmation within twenty-four hours. Notification of this ruling was sent to all physicians, midwives and hospitals. The Chicago plan provides: (1) twenty-four hour ambulance service by the city—a special ambulance equipped to meet emergency requirements is stationed at the Chicago Maternity Center; (2) premature ward care, with special equipment for oxygen therapy and other types of emergency therapy; (3) field nursing service for instruction of the mothers; (4) a supply of a simple type of heated bed, lent for the use of graduates in the home (this is of special value in reducing the number of return cases due to acute illnesses after discharge), and (5) an outpatient clinic maintained for instruction of mothers and the care and supervision of graduates not having private physicians. The board of health also provides breast milk free of charge from its Breast Milk Bureau.

In Chicago the mortality rate among all newborn infants in 1934, the year previous to the inauguration of the citywide program, was 47.7 per thousand live births, while in 1945 it had dropped to 29.6 per thousand in infants under 1 year of age. In 1935 the mortality rate

among 1,862 reported premature infants was 42.6 per cent. In 1945, of 3,716 reported premature infants (which represented 6.22 per cent of all newborn infants, totaling 59,343) the mortality rate was 21.7 per cent in the first two weeks of life and 30.7 per cent during the first year of life.

The conclusion which might be drawn is that the program established in Chicago for premature infants had an almost immediate effect on the lowering of mortality among all infants.

A further point to be emphasized is the striking reduction in morbidity among the premature infants received in the two stations, and this relates particularly to infections of the eyes, the skin, the respiratory system and the gastrointestinal tract.

In 1942, out of over 5,000 neonatal deaths in the state of Illinois (deaths during the first month of life) 35.5 per cent were certified as due to prematurity. Two special stations were opened downstate, one each in Peoria and Springfield. In 1943 and 1944 the mortality rate among almost 500 consecutive cases in these two stations was reduced to 17.3 per cent in 1943 and 24.1 per cent in 1944. The majority of deaths contributing to the higher figure in the 1944 series occurred in infants admitted from outlying areas to the center as its facilities became better known throughout the state, several days to even weeks after delivery and often in a moribund state. In order to protect the infants and assure early transportation from distant communities to the special nurseries, arrangements are in effect with private ambulance companies in Peoria and Springfield who on call send an ambulance to the station for premature infants to procure a portable incubator and a trained nurse prior to driving to the community where the premature infant has been born.

So much for statistics which might be related to the subject at hand, lowering and prevention of infections.

THE FUNCTION AND OPERATION OF THE PREMATURE STATION

A. As Related to the Nursery for Newborn Infants in a General Hospital.—As there are only a limited number of cities in the United States having a special children's hospital, in most cities the premature infant born in a general hospital will be cared for in the nursery for newborn infants.

An even more difficult problem must be solved when an infant is born in the home and it is to be admitted to a hospital nursery, as most hospitals regard such infants as requiring isolation care.

B. Special Station in General and Children's Hospitals.—The station for premature infants must fulfil, in greater detail, the minimum requirements outlined for the nursery for full term infants, and in addition must meet those needs that are peculiar to the premature infant. The minimum provisions should include: (1) a room adjacent to the

nursery proper set aside for such infants; (2) a separate room to provide for isolation care; (3) transfer of the infants to the larger nursery as they more nearly approach maturity, to prepare them for graduation so that they may become accustomed to a lower room temperature, and (4) at least one nurse experienced in the care and feeding of the premature infant on duty during each of the eight hour periods.

C. Prevention and Control of Infections as Related to the Pediatric Medical Staff.—A pediatrician should be in charge and have responsibility for the over-all conduct of the station; a resident physician or another member of the pediatric staff should serve with the former, and these men alone should assume the duties for carrying out the established regulations for the conduct of the station and the nursing and medical care during their periods of service. They and all physicians entering the station should carry out the established technics for gowns, caps and masks, and gloves if the latter are indicated. The internes are discouraged from entering the station except as they accompany the pediatrician and resident, or in an emergency.

The Supervising Nurse and her Staff: The place occupied by the nurse in the care of the premature infant is of major importance. A nurse should not choose "the care of the premature infant" as her elective after she finishes training unless she is sincerely interested in this branch of pediatrics.

The nursing standards must be carried out during the entire twenty-four hours. Technic and observations during the evening and night are as important as those during the day. Never can there be any relaxation of good technic, intelligent observations and efficient nursing care.

Since clinical observations are of more diagnostic value than the signs detected on physical examinations, conscientious observations are of the greatest importance, and her attention should be directed to every change in the infant's condition.

Nursing personnel must be impressed with the fact that the responsibility for the occurrence of infections in the nursery rests largely with them. They must constantly be alert to prevent breaks in technic. If student nurses are in the nursery, a supervisor should always be present so that their technic can constantly be checked.

Proper management of the ward for premature infants requires that the supervisor or her assistant inspect each premature infant during the bathing period. Nurses should be taught to watch the infant's color closely. The color is a most important indication of the condition of the baby. Pallor as well as cyanosis must be noted. Of equal importance are changes in respiration, abdominal distention and changes in stools. Since the infant's progress and welfare are so dependent on the nursing care, the nurse must be conscientious about reporting her discoveries to her supervisor and the physician in charge.

Unfortunately, in the organization of many nurseries, too great thought is given to elaborate equipment and not sufficient consideration to adequate nursing personnel. In many hospitals the executive officers do not hesitate to spend large sums of money for the finest of equipment but refuse to employ a sufficient number of nurses to permit aseptic technic. A shortage in the nursing personnel accounts for inadequate care, especially during the critical first hours of life, and may result in epidemics of diarrhea, impetigo and other types of infections among the infants.

For the twenty-four hour period, the nursing personnel should average one nurse for every 2 or 3 infants. The number of nurses required also depends on whether or not graduate or undergraduate nurses are in charge of these babies.

In hospital wards, the constant changing of nurses which occurs so frequently to meet the curriculum for nurses' training in general hospitals is found to be a great disadvantage. At the Sarah Morris Hospital for Children a satisfactory solution has been found for some of the nursing problems through the employment of "nursery maids" (infant nurses as a part of the personnel in the station for the premature infants). These young women, who are entered for one year's training in the care of newborn and young infants, spend part of their training period in the obstetric nursery and part in the children's hospital, in which the station for premature infants is located. Since they remain in the station for longer periods than the average nurse in training, the personnel is changed less frequently, which is a great advantage and decreases the amount of personnel instruction necessary.

Cooperation with the Obstetric Staff: In the conduct of the station for premature infants in a general hospital there should be the closest cooperation between obstetricians and pediatricians. Frequent consultations and interval rounds of the lying-in division and nursery should be made jointly by them. It is unfortunate that in many institutions, notwithstanding the fact that the visiting pediatrician is responsible in name, he does not assume the full requirements of his duties.

I take for granted that it is understood that I have been talking largely of service cases. It must be realized that so far as private cases are concerned the dividing of responsibility for the care of the infant is one of mutual consent.

The Consulting Staff: It has been my experience that often the views of the consulting staff are less conservative than those of the pediatrician. Once upon a time paracentesis of an ear drum was a common practice in the station of the Sarah Morris Hospital for Children—as against not one in the past several years. Spinal puncture has also become one of the rarer procedures since Jerome Glaser spent a year in this station in 1930 studying how and how not to do it and

the evaluation of practical results attained. Here again, confidence and understanding are necessary in the conduct of the individual case.

ISOLATION TECHNIC IN A NURSERY FOR YOUNG INFANTS

Dr. Iwan Rosenstern has recently reported on his experience of seven years with air-borne infections at The Cradle in Evanston, Ill. Their new building was erected in 1939. The station is air-conditioned and provides mechanical barriers and germicidal lights of various types. He reports the results in units equipped for air conditioning in which it was the only principle used to prevent air-borne infections. He found air conditioning alone to be unsatisfactory.

Much better results were obtained when germicidal irradiation through lights placed above the open end of the cubicles threw a vertical curtain of light across the entrance. The cubicles with barrier light curtains are so designed that only a harmless fraction of the rays reaches the corner of the cubicle where the head of the infant is exposed. In the presence of irradiation the nurses' eyes were protected by head shields and goggles.

An appreciable reduction in the number of respiratory infections as compared with those units in which air conditioning alone was used resulted. In units with the vertical curtain at the entrance 0.3 respiratory infection of infants per year was experienced. The cross infections were less than one twentieth of the number in air-conditioned control units without irradiation.

Doctor Rosenstern found a great variability in the efficiency of the various types of face masks used in his study.

Having described what might be termed an ideal series of units for isolation technic, I will briefly describe our station at Sarah Morris Hospital for Children.

Our station consists of three rooms, one for infants when first admitted, a second for infants being prepared for graduation and one used as an isolation unit. No attempt is made at air conditioning. The temperature is fairly well controlled and, weather permitting, windows are allowed to be kept open. For individual control of the temperature of the infants we depend on electrically heated beds, it being assumed that infants in their individual beds are in what might be termed cubicle isolation. Our infants are, therefore, dependent on the isolation technic as carried out by our nurses for the prevention of cross infections.

When the station was opened in 1922 one of the objectives was to demonstrate that a general hospital could provide good care for premature infants with a minimum of equipment if the nurses were skilled and conscientious in carrying out the regulations established for the protection of the infants.

After an epidemic of infectious diarrhea in a Chicago hospital some eight years ago the Chicago Board of Health required nurses to wear masks, caps and gowns in all nurseries for newborn infants. There was so much complaint on the part of our nurses who were working in a room with high temperature that at our request we were privileged as a control study under the board of health to discard the use of masks by the nurses. In the past seven years no masks have been worn by nurses in the station. Nurses in attendance with infections of the respiratory tract and other infections are excluded from the station.

It is our belief that in the presence of virus infection it is difficult to see how several layers of gauze will stop the virus when a Berkefeld filter will not. If the infection is bacterial the known inefficiency of most types of masks after a short period of time makes them more or less ineffective. If masks are to be worn a sufficient number must be supplied so that they can be changed as often as once in two hours. The pediatric medical staff, however, wears masks and gowns while visiting in the station.

ASEPTIC NURSING TECHNIC

Morbidity is always potential mortality. It is the responsibility of the hospital to prevent morbidity from occurring both in the premature and in the full term infant. We believe that one of the most important regulations is the thorough washing up to the elbows by the use of soap and running water (not scrubbing). The hands and arms should be washed on entering the nursery between the handling of each baby, after diapering, before feeding and after the use of a handkerchief or the adjusting or handling of a mask if one is being worn.

In the handling of the baby the upper part of the body may be considered as being the clean part and the lower half as being contaminated. The nurse is not allowed to handle the upper part of the bed or the baby after caring for the lower half without washing her hands.

These represent two important regulations for aseptic nursing technic as practiced in our station.

PREVENTION AND CONTROL OF SPECIAL DISEASES

Cutaneous Infections.—Miliaria, impetigo and staphylococcic and streptococcic infections are dangerous and invite rapidly spreading epidemics in the ward.

Impetigo: The recent report of Aldrich and Holmes [1] on the treatment of impetigo by intramuscular injections of penicillin is in line with our experience. They recommended two doses of 5,000 units of penicillin each with an interval of three hours between the doses.

1. Aldrich, C. A., and Holmes, C. A.: Impetigo, Am. J. Dis. Child. **72**:279 (Sept.) 1946.

We have used larger amounts more frequently repeated with good results. They expressed the belief that it is safe to return many infants to the ward within thirty-six hours. Our period in the isolation unit has been longer. It has also been our practice to open each vesicle thoroughly and apply a 10 per cent solution of silver nitrate. We also expose, as far as possible, the infected area to open air and ultraviolet radiation.

Infections of the Gastrointestinal Tract.—Infections of the intestinal tract rank in importance with infections of the respiratory tract. It is well known that infection of either of these systems is likely to run rampant throughout hospital wards unless the individual cases are properly segregated at the outset. It is not so many years ago, before our wards were properly equipped to care for premature infants, that doubt as to the advisability of instituting hospital treatment for young infants in preference to care in their homes, even though facilities for general care were limited, existed in our minds with good reason.

No subject with which physicians have to deal in the care of premature infants calls for such mature judgment as the care and treatment of gastrointestinal infections. In fact, the entire hospital unit is more or less planned and constructed with the idea of prevention and isolation of these cases. Thought should all be centered on their prevention, since once they become established their course is associated with the gravest dangers.

All intestinal disturbances must be considered serious because at the outset it is impossible to decide whether an intestinal disturbance is simple indigestion or the first symptoms of an infectious diarrhea. Again, it is rather difficult to determine whether a disturbance is caused by abnormal activities of the intestinal flora, pathogenic bacteria or unknown types of virus infection. I would call attention to the often times frequent stools seen at the end of the first week of life which follow the meconium stools in the first days of feeding with breast milk or cow's milk and which are not of serious consequence and do not impair the general health of the infant.

Time will not allow discussion of the supportive therapy, feeding and special medication for infants with infectious diarrhea.

Pneumonia.—Bronchitis during the first few days of life may be the result of aspiration of infectious material, or it may accompany a general septic infection. After birth, infection of the bronchi may reach the child from an infected mother or attendant. Infections in the upper air passages may spread directly to the deeper structure of the respiratory passages, and there produce bronchitis or bronchopneumonia.

The onset is most often insidious in the newborn infant and especially so in the premature infant. At first, a slight nasal discharge and a cough of varying severity may possibly be noted, although these symptoms

may be entirely absent in weak infants. Soon, increased frequency of respiration appears, accompanied by dilatation of the alae nasi. The rate of respiration increases to 60 or 80 per minute and often the infants are too weak to cough. Attacks of cyanosis occur, their severity in most cases corresponding to the severity of the pneumonia. The temperature may be only slightly elevated or even subnormal. There is a noticeable loss of weight, and the stools may become dyspeptic and greenish and contain mucus and undigested particles which may lead to an erroneous diagnosis. The prostration may be extreme, and the infants may become apathetic or stuporous. Abdominal distention is of frequent occurrence and constitutes a serious complication.

The severity of these early symptoms is due either to the sudden intense congestion of the small alveoli, which interferes with the bronchopulmonary apparatus almost as much as does consolidation, or to the intensity of the infection.

Treatment: Prophylaxis. The prevention of pneumonia in premature infants requires that these weak babies be protected against infection from every source. Any person suffering from an infection of the respiratory tract should be excluded from the nursery. If the premature infant is in the nursery for newborn infants, it should be isolated from the other babies. Only when the attendants are thoroughly trained in the principles of aseptic nursing is it safe to leave the infant in close proximity to others who have infections of the respiratory tract. All utensils should be individualized and should be sterilized before use.

As soon as these babies can be removed from their heated beds, they are to be kept in a large, well ventilated room which is not overheated. The babies should be given the benefit of open air and sunshine as soon as their development warrants.

General Treatment: The treatment of pneumonia is preeminently that of watchful expectancy. Overtreatment must be avoided, since these feeble infants are unable to withstand overmanipulation or stimulation. Generally, pneumonia in robust infants is an acute self-limited disease, but in premature infants the course is apt to be somewhat subacute without the tendency to limitation. The indications for treatment are to support the heart and avoid intestinal distention. The feeding problem of the premature infant is not easy at any time, but during an attack of pneumonia it becomes doubly difficult. Small feedings are advisable to aid in preventing distention, vomiting and diarrhea. Feeding by gavage is often desirable. However, this is not always possible, and it may be easier to feed the baby with a dropper or a small bottle, especially if much mucus accumulates.

The hygiene of pneumonia requires that the child receive plenty of fresh air. The one best therapeutic measure is the continuous administration of oxygen at the first suspicion of pneumonia. This

is most satisfactorily carried out in an oxygen chamber or by nasal catheter. The prime indications are for promotion of elimination and a sufficient administration of fluids. The routine use of drugs, such as cardiac and respiratory stimulants, is not favored. Whisky or brandy may be administered in quantities varying from 5 to 30 drops every three hours depending on the indications and the weight of the infant. Aromatic spirit of ammonia, in doses of 1 to 5 drops, is one of the most effective stimulants. Both the whisky and the ammonia should be given in the milk feedings, or well diluted in at least 8 parts of water. In cases of emergency, sudden heart failure or weakness accompanying a sudden drop in temperature, 1 or 2 minims (0.06 to 0.12 cc.) of epinephrine hydrochloride, 1 in 1,000 solution, or ½ grain (30 mg.) of caffeine and soduim benzoate to the dose, given hypodermically, will be found to be a quick acting, reliable cardiac and respiratory stimulant.

Frequent, regular changes of the infant's position in bed are imperative for the successful care of premature infants with pneumonia.

The specific treatment with sulfonamide compounds and penicillin, or a combination of them, should be carried out by the method prescribed for the full term baby with consideration of the fetal age and development of the infant and its body weight.

The subject of prevention and control of infection in the premature infant could be expounded indefinitely. From what has been stated covering care of the premature infant it must be realized that there is offered a field for practical and specific use of the more recent advances in hospital technics, engineering, physiology and serology, and biochemical and antibiotic therapy.

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MANAGEMENT OF EMERGENCIES

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PREMATURE infants as a group pass through their period of greatest danger in the first forty-eight hours of life—it is in this period that 90 per cent of the mortality among premature infants will be encountered.

Unfortunately the premature infant, having weathered this initial forty-eight hour danger zone, cannot now be promised smooth sailing for the remainder of his voyage. A premature infant must still navigate dangerous waters and be exposed to many more hazards than is his more fortunate full term compatriot.

Obviously our first medical responsibility is to try to get the premature infant through this forty-eight hour period of danger. It is now agreed that the lot of these infants cannot be improved by procedures that are instituted after birth. The obstetricians recognize that improvement in the condition of premature infants in the first forty-eight hours after birth can be achieved only through forces acting in the prenatal and natal periods.

EMERGENCIES ENCOUNTERED BEFORE BIRTH

In the ideal relationship between obstetrician and pediatrician the two work as a team. The first "emergency" situation arises when the obstetrician notifies the pediatrician that their patient is either starting spontaneous premature labor or in his opinion should be delivered prematurely because of certain complications of pregnancy that have arisen.

The pediatrician in this relationship is equipped by training and primary interest to act as the infant's advocate in representing the baby's best interests in the working out of this problem. The greatest danger which the fetus faces is that resulting from the effects of anoxia. Under certain conditions, as in hemorrhage from placenta previa, the danger from anoxia is so great that 80 per cent of the babies will die even though alive at birth. Further, in the presence of this type of severe anoxia, the fetus probably already has suffered so much cerebral damage that it would be unwise to expose the mother to any unnecessary hazard at delivery for the sake of a potentially damaged baby.

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Fortunately, in most situations in which premature delivery is impending the fetus can be assumed to be a good risk in good physical condition. Under these conditions the type of delivery and the type of anesthesia or analgesia can be elected with a view toward the protection of the fetus from trauma and anoxia. When obstetric conditions permit, the method of delivery most likely to produce a vigorous active premature infant in good condition is the delivery of the baby from below by episiotomy and low forceps without administration of any type of analgesic or anesthetic to the mother.

EMERGENCIES ENCOUNTERED AT THE MOMENT OF BIRTH

The delivery room should have certain supplies and equipment instantly available for the management of emergencies: (1) sterile, warmed, cotton blanket for premature infants; (2) sterile sponges; (3) heated crib; (4) mechanical suction with tracheal catheter; (5) oxygen tank with mechanism for measuring and controlling the amount and pressure of the gas and a suitable mask for infants, and (6) emergency tray with sterile syringes and needles, sterile ampules of alpha-lobeline, nikethamide and epinephrine hydrochloride.

As the obstetrician delivers the baby he clears the air passages by postural drainage and suction and then clamps and cuts the cord. At this point the obstetrician places the baby in the sterile blanket for premature infants held by the assistant assigned to care for the infant. The infant is placed in the heated crib, and the airways and mouth are again cleared by drainage and suction. If the baby has not begun to breathe the oxygen mask is placed over the baby's face and the heart is auscultated. If the heart rate is relatively normal and of good quality the baby is in no immediate danger, and one can wait a short time for the infant to breathe spontaneously. The heart is watched closely—if the rate begins to slow up some method of artificial resuscitation should be used, either a closed system or mouth to mouth breathing through a protective thickness of sterile gauze. With either method, following gentle inflation of the infant's lungs, gentle pressure on the infant's chest is used to deflate the lungs. The rate of this artificial respiration should not exceed 8 to 10 times per minute.

If under this regimen the heart rate picks up one can wait a further interval for spontaneous respiration to become established.

If, however, at any time the heart rate becomes alarmingly slow the infant's condition is precarious. In the presence of this real emergency the normal respiratory center in the medulla is unable to function and stimulation must be given the carotid and the aortic chemoreceptors if the infant is to survive. To accomplish this end I prefer to use 0.2 to 0.4 cc. of alpha-lobeline intravenously and to inject it into the longitudinal sinus or directly into the heart. Anyone who has had

experience with this procedure can testify to its dramatic and instantaneous effectiveness. The infant instantly starts gasping respiration, the heart rate picks up and the infant's color and tone improve.

If the infant is so injured that it cannot maintain this initiated respiration or if the carotid and aortic receptors are themselves so injured that they cannot respond, then, in my experience, further efforts to resuscitate the infant have been of no avail.

Once respiration has been established one should take a moment to appraise the baby's general condition. If the infant's color tone improves, a vigorous cry appears, the lungs become resonant, the chest shows no retraction of the costal margin and the respiration is free from grunting expiration, one can assume that the infant probably will do well so far as the respiratory system is concerned. The heart has already been examined, but one must wait until later for a final appraisal of its condition, for it is known that murmurs heard this soon after birth may disappear and be of no importance while serious murmurs may not be present at birth and may only appear later. Intrauterine anoxia of sufficient duration may have caused cerebral edema and hemorrhage with symptoms detectable at birth and may have been the main cause of the difficult resuscitation. On the other hand, anoxic injury may have been so recent that these changes may become clinically evident only a number of hours following birth. A gross intracranial hemorrhage, such as would result from a ruptured vein of Galen or from a torn major sinus, would cause fatal symptoms at birth; however, smaller degrees of hemorrhage require an appreciable period of time before clinical symptoms become evident.

Therefore at birth, even though the infant responds well to efforts at resuscitation or even if breathing has begun spontaneously, one must still be guarded in prognosis, at least until the infant has demonstrated his ability to survive the fateful first forty-eight hours in good condition.

The infant, still far from safe, is transferred to the nursery for premature infants as soon as his immediate condition permits.

EMERGENCIES ENCOUNTERED IN THE FIRST FORTY-EIGHT HOURS OF LIFE IN THE NURSERY FOR PREMATURE INFANTS

During the premature infant's first forty-eight hours of life in the nursery for premature infants my colleagues and I are particularly alert to detect evidence of anoxic or traumatic injury or of some congenital defect of development.

We assume that all prematurely born infants may well have suffered some degree of anoxia in utero, during delivery or during the resuscitation period. For this accomplished anoxic injury little can be done, but much can be done toward reducing or preventing further anoxic damage by placing the infant at once in an atmosphere of from 50 to

60 per cent oxygen. With us this procedure is routine for all premature infants no matter how good their condition seems to be on admission to the nursery.

Anoxia initiates a progression of changes in infants that can be recognized as congestion, edema and hemorrhage that involve any or all organs and tissues of the body. The newly arrived premature infant should be watched particularly for signs of developing edema and hemorrhage. These changes in the lungs produce increasing respiratory distress, and the baby's life will depend on enough oxygen-absorbing tissue's remaining so that, with the help of the added oxygen, the infant will be able to absorb enough to sustain life until the forces of recovery can set in. These changes in the central nervous system may result in paralysis of the respiratory center. The infant must be able to absorb enough oxygen to sustain the respiratory center and to prevent the development of more cerebral congestion, edema and hemorrhage. It is possible at times to relieve some of the symptoms resulting from increased intracranial pressure through repeated lumbar punctures.

It is probable that the incidence of congenital defects is higher in premature infants than in infants born at full term. Premature infants with excessive mucus with or without cyanosis are immediately suspected of having a tracheoesophageal fistula until this possibility is excluded. Infants with vomiting are suspected of having some form of intestinal obstruction until this possibility is excluded. We have seen premature infants with malrotation of the cecum with obstruction due to a short mesentery's crossing the second portion of the duodenum, with intrinsic atresias, with pyloric stenosis and with diaphragmatic hernias. The occurrence of these conditions represents a real emergency, for the infant's size is no contraindication to operation and the entire prognosis rests on early operation made possible by an early diagnosis.

During the first forty-eight hours of life we therefore place all premature infants in an atmosphere of oxygen and maintain the incubator at a temperature that allows the baby to stabilize his body temperatures between 96 and 98 F. The infants are given absolutely nothing by mouth during this forty-eight hour interval, and they are watched constantly, twenty-four hours a day, by trained nursing personnel, with trained pediatric personnel in residence and on instant call.

EMERGENCIES ENCOUNTERED AFTER THE FIRST FORTY-EIGHT HOURS OF LIFE

The Edematous Baby.—The management of the premature infant showing varying degrees of subcutaneous edema is one of the more important problems encountered. We assume that this edema, which at times may approach generalized anasarca, is the result of preceding

anoxic damage to the capillaries, allowing the escape of fluid into the tissues. Bitter experience has shown that too early administration of fluids to these babies causes increased edema. If this increased accumulation of fluid were confined to the subcutaneous area little damage would result, but when the increased edema fluid involves the lungs or the central nervous system death may quickly ensue with appropriate symptoms.

We have been convinced on purely clinical grounds that infants with edema should not receive fluid enterally or parenterally until they have themselves absorbed their subcutaneous fluid even though this means the withholding of fluids for four or five days from even the smallest babies.

The Atelectatic Baby.—The premature infant with massive atelectasis shows pronounced respiratory distress, retraction of the costal margin and even collapse of the chest wall until the pericardium can be seen rounding out the thorax. The treatment of atelectasis consists of placing the infant in an atmosphere of 50 or 60 per cent oxygen.

If the infant is fortunate and there is sufficient alveolar duct and alveolar absorption of oxygen to keep the baby's color pink while he is in the oxygen tent, even though he becomes cyanotic outside, there is a good chance that the infant can be sustained until the lungs begin to open up. Again, experience has shown that the infant atelectatic lung rather regularly begins to expand between the third and the fourth day of life. This is apparent clinically by inspection, auscultation and palpation and by roentgen ray.

Again, since we assume that the atelectasis is the result of anoxia and that congestion, edema and hemorrhage are responsible for much of the pulmonary changes, fluids should not be given by mouth at any time during the first three days and parenterally only when the infant's skin turgor suggests the need for fluids.

The Cyanotic Infant.—The cause of cyanosis in a premature infant is usually found to be pathologic changes in the lungs, the brain or the heart. Frequently the true cause is arrived at only by the process of exclusion.

Pulmonary changes sufficient to produce cyanosis are readily detected by percussion and confirmed by roentgenologic examination.

Congenital heart disease may or may not be accompanied by a murmur, but a congenital cardiac lesion sufficient to cause cyanosis will usually be accompanied by hypertrophy evident on roentgenologic examination.

Cerebral edema or hemorrhage occasionally may be suspected from the examination of the anterior fontanel, but unfortunately disease located below the tentorium may not make itself evident through the

fontanel until too late to be of much help. We believe that in case of doubt, lumbar punctures should be performed freely. Not only will the finding of clear fluid under excessive pressure, xanthrochromic fluid with or without large numbers of crenated red blood cells and accompanied by a high protein level, moderate amounts of old and fresh blood or grossly hemorrhagic fluid make the diagnosis obvious, but the removal of such fluids will exert a beneficial therapeutic effect. It is our practice to repeat lumbar punctures on the recurrence of symptoms but also every two or three days until the character of the fluid returns to the normal range.

The Apneic Infant.—It is not at all uncommon to encounter a premature infant whose prior course has been uneventful who suddenly just stops breathing. All who have been present during one of these episodes are convinced that if the episode had passed undetected or if stimulants and artificial resuscitation had not been practiced the infant would have quietly died. This syndrome is almost invariably accompanied by the finding of grossly abnormal fluid on lumbar puncture. The fluid is usually xanthrochromic, containing crenated red blood cells on microscopic examination and having a high protein content. The withdrawal of this apparently irritating fluid is accompanied by at least temporary relief of symptoms. With recurrence of symptoms a second tap again gives relief.

The Convulsing Infant.—The premature infant with convulsions is treated at once by lumbar puncture. The fluid is again carefully studied in an effort to arrive at an accurate diagnosis. While most convulsions are the result of anoxic or traumatic damage to the central nervous system, it must not be forgotten that infection may be the cause. All fluids obtained by lumbar puncture should be cultured and smeared for organisms. We have seen 2 cases of meningococcic meningitis contracted in utero within a year.

If the fluid obtained by lumbar puncture is normal, no examination is complete until subdural hematomas are excluded by subdural taps.

The Vomiting Infant.—In the premature infant who vomits, conditions included in differential diagnosis are congenital abnormalities, intracranial pathologic changes, infection and faulty feeding technic. Many premature infants with unexplained vomiting are found to have grossly abnormal spinal fluid under pressure, and too often to be coincidence their symptoms disappear with the withdrawal of this fliud.

Many infants who regurgitate or vomit may aspirate food or stomach contents and contract a chronic pulmonary infection on this basis. Associated with this infection, vomiting may become more pronounced and other clinical signs of pulmonary infection may appear. The aspiration may be sudden, acute and result in death and be dis-

covered only at postmortem examination. Constant observation and, when this disaster occurs, immediate treatment by drainage and suction are demanded if a catastrophe is to be prevented. We believe that the possiblity of aspiration is minimized if a small infant is fed small amounts every two or three hours by gavage feedings.

The dangers of aspiration of oil through the administration of oily vitamin preparations is so real that we recommend the use of water-soluble vitamin preparations for premature infants.

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FEEDING OF PREMATURE INFANTS

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THE FEEDING of premature infants in the hospital nursery will be discussed under three headings: (*a*) problems during the first week of life, (*b*) problems after the first week and (*c*) problems on discharge to the home.

PROBLEMS DURING THE FIRST WEEK OF LIFE

During the first week, while the infant is making numerous physiologic adjustments incident to birth, the problem is to meet his minimum maintenance food requirements without exceeding his ability to ingest and retain the foodstuffs offered. There are, then, two considerations: technic of feeding and a correct estimate of maintenance requirements.

Technic of Feeding.—Major emphasis must be placed, during this period, on the proper choice of the method of feeding, i. e., from nipple, medicine dropper or gavage, whichever is indicated by the individual infant's strength. The quality and the quantity of nursing care will also help determine the choice of the method of feeding, and this in turn will define the interval and the amounts to be fed. If nurses are available who are expert in the use of gavage, larger individual feedings can be offered at longer intervals. If such nursing service is not available it may be necessary to give smaller amounts at more frequent intervals, for example, every two hours by medicine dropper. If gavage is to be used it must be done expertly. Although the crude danger of pouring fluid into the trachea is easily prevented, it must be remembered that reflex attacks of apnea and circulatory collapse can be produced by excessive handling.

Careful nursing records should be kept of the infant's feeding behavior so that indicated changes may be made promptly by the physician in charge. One of the chief dangers is that the feeding of too large amounts may lead to regurgitation or vomiting with aspiration, or to abdominal distention, with serious implications for the infant. If any of these symptoms occur they need to be called to the attention of the physician in charge immediately.

Maintenance Nutritional Requirements.—Requirements for energy and fluid are low during the first week. The total energy requirements

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are low because (1) the basal metabolism at this time is only from 40 to 50 calories per kilogram per day, (2) the expenditure of energy due to activity is minimal, (3) the quotas for fecal loss and specific dynamic action which depend on the food intake are small and (4) no calories need be supplied for growth. It is obvious that the total negative energy balance will be small even if the caloric intake be zero. Fluid requirements are also relatively small because fluid needs to be supplied only for maintenance and because, with a low expenditure of energy and the minimal need for sweating there will be relatively little loss through skin and lungs. Furthermore, initial stores of body water usually permit some delay in the administration of fluid. In most infants, then, after the first twenty-four hours, the administration of approximately 100 to 120 cc. per kilogram will probably be adequate, provided that there is no vomiting.

Initial Feedings Every Three Hours for Infant of 1,500 Gm.

Age	5 per Cent Glucose, Cc./Feeding	Feeding Mixture, Cc./Feeding	Total Fluid Cc./Kg./ 24 Hr.	Total Calories, Kg./24 Hr.
0-12 hr.	0	0		
12-24 hr.	8	0	20	..
24-36 hr.	8	4		
36-48 hr.	8	8	75	30
	Water			
3, 4, 5 days	8	12-16	105-135	50-70
6, 7 days	5	20-25	125-160	85-105
8 days	0	30	160	125

The schedule of initial feedings in table 1 illustrates a method of meeting the requirements by a gradually increasing food intake. The feeding mixture used consists of 18 Gm. of a powdered preparation of cow's milk from which half the cream has been removed and 11 Gm. of carbohydrate with water added to 150 cc. This mixture contains 0.8 calory per cubic centimeter. It is a feeding high in protein, high in carbohydrate and low in fat and protein supplies 20 per cent of the calories, fat 16 per cent and carbohydrate 64 per cent. The schedule has been prepared for an infant whose birth weight is 1,500 Gm. and who is able because of his own vigor to go on a three hour feeding schedule or for whom adequate nursing service permits the use of gavage every three hours. It is seen that during the first twelve hours the infant is given nothing by mouth and that for the second twelve hours he is given 8 cc. of 5 per cent glucose every three hours. During the first twenty-four hours no particular attention has been paid to either his total fluid or total caloric requirements. It is assumed that he will not starve to death and that his initial stores of body water will prevent dehydration. If diminished tension of the fontanel and elasticity of

the skin indicate dehydration it may be desirable to give him fluid parenterally such as lactate-saline solution subcutaneously.

If the infant has ingested and retained the amounts offered on the first day, then for the third twelve hours of life he is given 8 cc. of 5 per cent glucose plus 4 cc. of the feeding mixture, a total of 12 cc. every three hours. During the fourth twelve hours of life he is given 8 cc. of 5 per cent glucose plus 8 cc. of the feeding mixture. It is seen that for the second twenty-four hours he will have obtained a total of 75 cc. of fluid and 30 calories per kilogram. On the third, fourth and fifth days 12, 14 and 16 cc., respectively, of the feeding mixture are given with 8 cc. of water every three hours. His fluid intake is now above 100 cc. per kilogram per day and is gradually being increased. His daily caloric intake rises to 70 calories per kilogram, maintenance requirements, by the fifth day. The administration of gradually increasing amounts of the feeding mixture leads to the ingestion of adequate fluid and calories for growth by the eighth day.

It is important that a schedule such as this be used only as a base from which one individualizes the feeding of each infant according to his ability to ingest and retain the amounts offered. For infants of less than 1,500 Gm. it may be important to offer less than proportionately smaller amounts. For example, an infant of 1,200 Gm. might be considered as entitled on a weight basis to 80 per cent of the amounts offered. Actually, he may tolerate only half of the amounts indicated. Conversely, larger infants may be able to take more than proportionately greater amounts. In the case of very small infants, below 1,000 Gm., it may be advisable to give nothing by mouth for the first twenty-four, forty-eight or even seventy-two hours and to make up the fluid requirements of approximately 100 cc. per kilogram per day wholly by the parenteral route.

Because of the tendency of premature infants to spontaneous hemorrhage 2 mg. of vitamin K should be given parenterally as soon as the infant is born, and this dosage should be repeated after forty-eight or seventy-two hours if the infant is not doing well. Because premature infants are born with low stores of vitamin C it is also probably advisable to give ascorbic acid during the first days of life. For the first two or three days 100 mg. may be given daily, either parenterally or orally. If ascorbic acid is given subcutaneously or intramuscularly it is important to use a properly buffered preparation to avoid an acid burn.

PROBLEMS AFTER THE FIRST WEEK

After the first week of life, when the more obvious physiologic adjustments have been made, the problem is to supply sufficient food for growth. Previous laboratory investigations have indicated that the premature infant has certain peculiar needs conditioned particularly by

defects in storage of nitrogen, calcium and phosphorus, by a defect in his ability to absorb fat and by his rapid rate of growth. These studies indicate the need for a relatively concentrated food of moderate total calories high in protein, calcium and phosphorus and low in fat.

Human milk, which has on the basis of clinical impression been considered the food of choice, is relatively dilute, low in protein, calcium and phosphorus and high in fat. The feeding mixture previously outlined, consisting of powdered half-skimmed milk diluted with water and reenforced with carbohydrate, meets the indications of the metabolic studies and has the added advantage that it can be concentrated according to the infant's capacity to ingest and retain the volumes offered.

The successful use for over twenty years of such a feeding mixture at the New Haven Hospital affords clinical evidence of the validity of this suggestion. At the New York Hospital, 14 infants whose weights on admission to the nursery were less than 1,000 Gm. have gained satisfactorily on this feeding. Furthermore, a study has recently been completed at the New York Hospital in which under constant environmental conditions and with the same medical and nursing care, 122 premature infants whose birth weights were between 1,000 and 2,000 Gm. were fed isocaloric amounts of human milk, a simple evaporated milk mixture or the partially skimmed cow's milk mixture which has been described. All the infants were given 25 or 50 mg. of ascorbic acid, 3,000 to 5,000 units of vitamin D and 20,000 to 25,000 units of vitamin A daily. For the period from the seventh to the twenty-eighth day, the mean daily weight gains per kilogram were as follows: for human milk, 12.5 Gm.; for the evaporated milk mixture, 14.1 Gm., and for the half-skimmed milk mixture, 15.7 Gm. The differences between the weight gains were all statistically significant. The infants were divided into two groups according to the mean birth weight (1,621 Gm.), and an analysis was made comparing the weight gains on the three feeding mixtures for the "smaller" and the "larger" infants. For the smaller infants, the mean weight gains were 11.7 Gm. on human milk, 14.9 Gm. on evaporated milk and 17.3 Gm. on the partially skimmed milk. The advantages of the cow's milk feeding and particularly of the partially skimmed milk were even more striking for the smaller infants in whom the metabolic peculiarities mentioned might be presumably greater. There can be no doubt that under conditions of modern hospital practice, with cleanliness, good refrigeration and careful supervision of feeding, the use of a partially skimmed cow's milk mixture will produce more satisfactory weight gains.

For the larger premature infants, however, there were no significant differences between the weight gains on the different feedings. For those infants whose vigor is such that one may reasonably expect to be able to put them to breast not too long after birth, every effort should therefore be made to maintain the supply of mothers' milk. This is

particularly indicated because of the problems that arise on discharging the infant from the hospital.

PROBLEMS ON DISCHARGE FROM THE HOSPITAL

The unexpected birth of the infant, possible illness of the mother at the time of the delivery, the precarious initial condition of the infant and the long period during which the infant requires highly specialized nursing and medical care and during which the mother is separated from the baby all combine to build up anxiety about his future course. Special measures need to be taken, beginning shortly after the birth of the infant, to mitigate this. Maintenance of breast milk so that the infant may be put to breast as soon as his vigor permits (a practical possibility in the case of larger infants), visits by physician and social worker to the mother while she is in the hospital, special instruction on the care of the infant as soon as mother and infant are strong enough and visits to the home by public health nurses both just prior to and after discharge of the infant to help the mother prepare the home will all contribute to more satisfactory emotional adjustment and feeding behavior.

I have discussed some of the factors which may guide one in the individualized feeding of premature infants. I should like now to present data on our experiences at the Colorado General Hospital during the last three months of 1946. The relatively small size of the service, the proximity of the nurseries for premature and full term infants, the interest of obstetricians, nurses, pediatricians and social workers in the problem and the cooperation of the Denver Visiting Nurse Association have permitted a choice of feeding best suited to the individual infant. Infants whose birth weights were less than 4½ pounds (2,041 Gm.) have been routinely admitted to the special nursery for premature infants and fed according to the system outlined. Infants who weighed more than 4½ pounds at birth and who were vigorous were admitted to the nurseries for full term infants and whenever possible sent to breast. The latter decision has been made on the basis of information supplied at the pediatrician's daily rounds by the obstetric nurses and the social worker concerning the personal factors which might make breast feeding a practical and desirable possibility. This procedure has been followed on the general assumption that the larger infants who are vigorous enough to nurse will not have pronounced metabolic handicaps which require the use of the partially skimmed cow's milk mixture.

During a three month period, 28 infants were admitted to the service for premature infants, with 2 deaths on the first day of life because of intracranial hemorrhage. Of the 26 infants who were discharged alive, 14, all in the larger weight group, were discharged on the breast at an average age and weight on discharge of 6 days and 5.1 pounds (2,313 Gm.) respectively. Follow-up data for a period of one to four months

after discharge have been obtained for 12 of the 14 infants. None had experienced acute illnesses, although there was a considerable incidence of disease of the respiratory tract in Denver at the time. This early discharge is not recommended for general use but does indicate what can be done with proper use of social service and visiting nurse facilities.

In summary, the feeding of each premature infant needs to be individualized on the basis of the facts available concerning his physiologic handicaps, his vigor and the services available for his care.

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DISCUSSION

Dr. Samuel Z. Levine, New York, Chairman: There are some controversial questions that have arisen here, but I think that if we were to sit around a table and talk them over many of them could be resolved. To make the discussion a little livelier we have asked for some formal discussion, and the first discusser will be Dr. Miner C. Hill, of New York, the father of the Mother's Milk Bureau.

Dr. Miner C. Hill, New York: The thorough, painstaking study of the physiologic handicaps of the premature infant by Dr. Levine and Dr. Gordon has added greatly to our knowledge of the premature infant's needs and how to care for them. I am in full accord with the environmental and supportive measures that their studies suggest, but I think that there should be greater emphasis on not translating the results therein obtained directly to full term infants and premature infants vigorous enough to nurse either from the bottle or from the breast. For the latter group even Dr. Gordon will agree that breast milk is preferable. To protect premature infants against infection they are not allowed to leave the nursery until they weigh 5 pounds (2,268 Gm.), and by that time the mother's milk has dried up. What are the criteria that determine how long a premature infant should be kept on the skimmed milk formula?

As a proponent of human milk as the initial food for all babies I would like to emphasize Dr. Levine's own summation. After balancing the pros and cons of mother's milk and cow's milk he concluded: "I should like to close this digression on the relative merits of human and cow's milk as the ideal food for premature infants by suggesting that in hospital practice or when human milk is not available except at great expense, a cow's milk mixture low in fat is an adequate substitute."

We must keep in mind that this work was done under ideal conditions, but is it practical for general application when only 10 per cent of the nation's babies are under the care of pediatricians? What about the premature infants in isolated rural districts, where physicians are not experienced in manipulating artificial formulas? They need consideration, too. The advance in the science of artificial feeding has done much to reduce infant mortality as a whole but has had little effect on neonatal mortality. That still accounts for 60 per cent of the deaths during the first year. I think that more study should be given to modifying breast milk for premature infants before resorting to artificial food. Modifying human milk is necessary during the first week or two, but we must keep in mind that breast milk is the only standard food that can be used throughout infancy without modification, simply by increasing the amount as the baby grows. Cow's milk was meant for calves and human milk for babies.

Dr. Gordon says of his experiment, "The results indicate that for smaller premature infants cow's milk mixtures, more particularly, partially skimmed milk, produce more satisfactory gains than does unmodified human milk."

I should like to ask Dr. Gordon some questions. Was this experiment controlled at the same time by an equal number of babies of comparable weight under the same environmental and nursing care fed on modified breast milk? What other procedures were employed, such as parenteral administration of isotonic solution of sodium chloride and plasma and blood transfusions? How often was it necessary to increase the formula or juggle the constituents? Among hospital premature infants I have observed that little effort is made to develop and maintain the mother's milk supply. How many of the 122 babies were put on breast feeding on discharge from the hospital?

Dr. Gordon reports that 14 infants under 1,000 Gm. gained satisfactorily on skimmed milk formulas. I am personally familiar with 5 premature babies who weighed less than 1,000 Gm. One was fed "alacta." One was fed 4 parts breast milk and 1 part evaporated milk. This baby was cared for at home, without an incubator. Two were fed foster breast milk. One was fed canned breast milk. They all maintained normal temperature, normal digestion and satisfactory gains.

The disturbing part of reports like Dr. Gordon's is that they get lay publicity and discourage mothers and physicians from giving breast milk first chance.

There has been considerable concern on the part of the Academy's Committee on Mother's Milk over a growing tendency among obstetricians, pediatricians and nurses taking care of the newborn to discourage breast feeding.

Just as light is produced in the incandescent bulb by resistance I think that light can be thrown on this problem by friendly disagreement. We are all working for the same end; the reduction of the neonatal death rate.

DR. HARRY H. GORDON, Denver: Dr. Hill, we are just as much concerned as you that obstetricians, pediatricians and nurses do not make more effort to have full term babies discharged from the hospital on breast feeding. It is important to remember, however, that many premature babies are too weak to nurse from their mothers. For these babies, the choice is not between breast feeding and artificial feeding. These babies will all be fed artificially because of their defect in nursing ability, whether cow's milk or human milk is used. Our data support the use of cow's milk. The feeding of pooled mothers' milk which has been pasteurized, frozen, stored, thawed and then heated prior to administration by gavage is just as artificial as the feeding of cow's milk.

You ask how long premature babies are kept on the half-skimmed milk mixture. It has been our custom to keep them on this feeding until they reach the weight of 5 pounds, and then we change over to a simple evaporated milk feeding. In the case of small infants, no attempt has been made to keep up the mother's milk supply. When the infant reaches the weight of 5 pounds, we change him over to an evaporated milk feeding mixture of 120 calories per kilogram, and if he gains on it, we send him home. If he fails to gain, we consider this evidence of a continuing defect in fat absorption and put him back on the partially skimmed milk mixture. The point which you have made, that breast milk should be used because it is subject to less manipulation, does not appeal to me as an argument in favor of the continued use of breast milk. I believe that it is our job to teach physicians how to use cow's milk and not to teach them to use breast milk in the feeding of small premature infants when there is so much evidence that its use is not advantageous.

The system which is now being used at the Colorado General Hospital and which has been described can work successfully only if we have the cooperation of obstetrician, pediatrician, obstetric, nursery, visiting nurses and the social service department. At pediatrician's rounds, both obstetric and nursery nurses and social

workers have attended. We have a small service, but we have been able to work out the system described.

DR. S. Z. LEVINE, New York: It seems to me that there is no fundamental difference in the thinking of the two discussers. I think that it should be emphasized on the basis of psychologic, bacteriologic and economic reasons that breast feeding is the ideal food for babies when they can take the milk directly from the breast. When breast milk is manipulated so as to become as artificial as cow's milk in feeding, then I think that the question should be raised whether we are feeding natural milk. The other question of modifying human milk falls into the same category. If one assumes that human milk is difficult to get, is more expensive and does not have a proved beneficial influence on the premature infant, why go to the trouble of modifying human milk when modified cow's milk serves admirably?

QUESTION: Dr. Clifford, might not spinal puncture in cases of cerebral hemorrhage naturally increase bleeding through lessening the intracranial pressure? Dr. Winthrop Phelps, in Baltimore, advises absolute rest for cerebral hemorrhages in infants, while some neurosurgeons, such as Dr. Charles Bagley, require daily lumbar taps. Why do you subscribe to the latter form of management of head injury?

DR. STEWART H. CLIFFORD, Brookline, Mass.: This question invariably comes up when this subject is discussed. If there were a rupture of a large vessel, such as the vein of Galen or a large sinus, the blood might conceivably pour out until stopped by the internal dynamics of the skull—under these conditions withdrawal of blood by lumbar puncture might cause fresh bleeding. However, this type of hemorrhage is fatal irrespective of the treatment selected. It is not this extensive type of hemorrhage that I am discussing. I am concerned with treating the patients with less extensive hemorrhage, usually the result of petechial hemorrhages or tears involving minor vessels. I believe that the treatment of this type of intracranial hemorrhage is by repeated lumbar puncture until the fluid clears. In my experience this method of treatment has not initiated fresh bleeding.

DR. SAMUEL Z. LEVINE, New York: Apparently in Dr. Clifford's experience repeated lumbar punctures do not increase the tendency to bleed intracerebrally. I think that the question, however, is still one that must be considered in the treatment of intracranial hemorrhage, particularly in premature babies. Lumbar puncture is an added burden which one must think about before subjecting these premature infants to repeated spinal taps.

QUESTION: How essential is it to separate the premature infants from the full term infants in the nursery, and how essential to separate those born on the outside from those born on the inside?

DR. JULIUS H. HESS, Chicago: I should like to convey the thought that the important factor in caring for the premature infant is the well trained nurse. The physician plays a less important part except in emergency. There is no reason at all why any general hospital cannot take care of premature infants in the nursery for newborn infants. I am talking of babies born in the hospital that are supposed to be clean from the standpoint of obstetric asepsis. Concerning babies that come in from the outside, most nurseries will not take them. If there is a room available where such an infant can be isolated for seventy-two hours, there is no reason thereafter why it cannot be removed and placed in the nursery if no complications have developed. With the use of sulfonamide compounds and the antibiotics when indicated, it is also usually safe after seventy-two hours' observation to put the babies in the nursery for newborn infants.

QUESTION: Dr. Gordon, what half-skimmed powdered milk do you use and what carbohydrate do you use?

DR. HARRY H. GORDON, Denver: We have used "alacta," a preparation of Mead Johnson & Company, but "Dryco," which is prepared by the Borden Company, has essentially the same organic composition. A preparation of powdered half-skimmed lactic milk may also be used. The carbohydrate which we have used is a mixture of dextrins and maltose.

QUESTION: Do you have any statistics showing that vitamin K is useful in hemorrhagic disease of the newborn?

DR. STEWART H. CLIFFORD, Brookline, Mass.: When the subject was brought up a good many years ago at a round table in this academy, figures were presented from the Boston Lying-in Hospital showing that if we were all agreed on our definition of what we call hemorrhagic disease there was no change in the incidence in hemorrhagic disease before and after the prophylactic use of vitamin K. Subsequently, similar observations have been pointed out by Dr. Potter, of Chicago. She reported a series of 5,000 deliveries in which vitamin K was used and a subsequent series of 5,000 in which it was not used. The findings in the two series showed no significant difference in the incidence of hemorrhagic disease. We have seen newborn infants with true idiopathic disease in cases in which both the mother during labor and the baby at birth received vitamin K. This is a controversial subject and I do not think that we have any right not to be open-minded as to the usefulness of vitamin K until the contrary is proved. Until we have more information we should continue to give vitamin K as at present.

QUESTION: Does germicidal irradiation predispose to retrolental fibroplasia?

DR. SAMUEL Z. LEVINE, New York: Retrolental fibroplasia is a condition in which there is an opacity behind the lens. It occurs in small premature babies weighing under 1,500 Gm. One theory is that light postnatally, particularly bright light, predisposes to the development of the condition. In many nurseries where there are control units without ultraviolet radiation and units where ultraviolet radiation exists, the incidence of retrolental fibroplasia is no greater in the units with ultraviolet radiation. Dr. Terry told me just before he died that he did not believe that bright lights predispose to retrolental fibroplasia.

DR. JULIUS H. HESS: Dr. Terry appeared in Chicago and asked for cases to demonstrate. I thought I had one and sent it to him and he made a diagnosis of congenital cataract. We use a good deal of ultraviolet radiation in our hospital and I could not find a single case. We may have missed some. More recently on inquiry among my colleagues I located 3 cases.

DR. SAMUEL Z. LEVINE, New York: It is interesting that the condition occurs in such varied fashion in different institutions. In Boston, Dr. Clifford once reported 12 per cent. In the New York Hospital in a series of 1,200 or 1,300 premature babies we have had only 3 cases. Apparently in Chicago the incidence is low.

QUESTION: Do you attempt to keep breast milk up by pumping the breasts of mothers of small premature infants (under 4 pounds [1,814 Gm.]) in hopes of putting the baby to the breast later?

DR. HARRY H. GORDON, Denver: No, we do not because we have not been successful.

DR. JULIUS H. HESS, Chicago: We do and are successful. It requires a will to cooperate on the part of the mother and salesmanship on the part of the physician and nurse in charge.

QUESTION: Do you feed sodium *r*-lactate one-sixth molar prophylactically?

DR. HARRY H. GORDON, Denver: We do not. When we need to make up the fluid intake we do not use isotonic solution of sodium chloride. We use a mixture of one third sodium *r*-lactate one-sixth molar and two thirds isotonic solution of sodium chloride.

QUESTION: Do you have acidosis developing in 2 to 3 week old premature infants? Give your opinion of feeding plasma in the first few days.

DR. STEWART H. CLIFFORD, Brookline, Mass.: Acidosis may develop in premature infants at any age. I recently saw a premature baby who had suddenly shown the typical picture of acidosis with gasping respiration and a lowered carbon dioxide level in the blood. I am convinced that this baby would have died if the house officer on duty had not recognized the condition and corrected the acidosis through the administration of sodium *r*-lactate one-sixth molar. It has been reported that acidosis is a frequent accompaniment of diarrhea in the newborn. We have found signs of acidosis even before the diarrhea develops to any degree of severity, so that it is almost a symptom that is present at the onset of the diarrhea. Acidosis is a real problem in premature infants, and one has to be on watch for it.

With regard to the use of plasma, we have not fed plasma to premature infants. We gave plasma intravenously for a while in an effort to build up the serum protein and to combat edema but found the amount which we gave had no effect in raising the level of serum protein.

DR. SAMUEL Z. LEVINE, New York: Again we profess ignorance. I do not think that any of us have used plasma routinely. It is being used with reported success, but we have not had personal experience.

QUESTION: The following procedures are recommended by the obstetricians. I do not approve, how do you feel? (*a*) One per cent aqueous solution of gentian violet medicinal is administered orally to prevent thrush. This is done in the delivery room. (*b*) One per cent ammoniated mercury ointment is applied to skin to prevent impetigo.

DR. JULIUS H. HESS, Chicago: Surprisingly enough, we see little thrush. When we see it we use bicarbonate of soda, and we use nothing prophylactically. I think that 1 per cent of gentian violet or even 2 per cent aqueous solution is perfectly safe. In our station for premature infants we use nothing on the baby except soap and water, for cleansing. If the skin is dry we may use one or two applications of warm mineral oil. We use no vegetable oils and no ammoniated mercury. Recently the pediatric department has persuaded the obstetricians to stop using ammoniated mercury in the nursery for newborn infants. There they had many cutaneous eruptions—not impetigo—and we believe that the ammoniated mercury was the causative factor in many of the cases.

QUESTION: What dose and how often during the day do you advise clysis for premature infants too small to take sufficient fluid by mouth?

DR. HARRY H. GORDON, Denver: We have said that we think that the baby should have between 100 and 120 cc. total fluid per kilogram as a minimum maintenance requirement. If he has taken 60 cc. we make up the difference by two clyses. We never give more than 40 cc. per kilogram of body weight in any clysis. The total amount will depend on how much the baby has ingested and retained.

QUESTION: In the case of edematous babies, do you withhold all fluids, including the formula?

DR. STEWART H. CLIFFORD, Brookline, Mass.: I am glad that this question was asked because it gives me an opportunity briefly to correlate our theory with that mentioned and presented by Dr. Gordon. I do not think that there is any disagreement between the technic Dr. Gordon presented and that which we practice. Please remember that the subject I was given to discuss was the treatment in emergencies, and naturally the babies so treated were sick infants. They were the abnormal babies that Dr. Gordon eliminated in his discussion. I have little doubt that if you confronted any one of us with an individual premature baby we each would come to the same conclusion as to the time when administration of fluids should begin. A baby that has been severely asphyxiated, who shows generalized edema, who exhibits poor muscular tone, poor reflex activity and considerable atelectasis—I think that we would all agree—is in no condition at that moment to begin taking food or fluids by mouth. We would wait until the infant's condition improved before attempting routine feedings.

DR. HARRY H. GORDON, Denver: I should like to say one thing about edema in premature babies. There are other factors leading to the development of edema in addition to the amount of fluid given. Occasionally there may be edema of the lower extremities at a time when there is actually hemoconcentration and a depression of the fontanel. The presence of edema of the lower extremities is not necessarily a contraindication to the parenteral administration of fluids.

QUESTION: How much alpha-lobeline do you give?

DR. STEWART H. CLIFFORD, Brookline, Mass.: We have used from 0.4 to 0.6 cc. of alpha-lobeline intravenously. I have not found the infants depressed by these doses. I might easily have missed noting a depressant effect that I could attribute to the drug, as it was not used except as a last resort in resuscitation, at which point the infant was already greatly depressed and almost moribund from anoxia. We gave up the use of "metrazol" (pentamethylenetetrazol) because of the convulsions which it produced.

QUESTION: As a general rule how much does a premature infant weigh at discharge?

DR. JULIUS H. HESS, Chicago: We try to discharge all those who are physically well off at 4½ pounds (2,041 Gm.) or less. We used to keep them until they weighed 5 pounds, but with the shortage of space we cannot do it. We can handle only 32 infants at a time. We try to get them out early if the home justifies it. We have a good follow-up service.

Abstracts from Current Literature

Biochemistry; Bacteriology, and Pathology

ACQUIRED RESISTANCE OF STAPHYLOCOCCI TO THE ACTION OF PENICILLIN. E. A. NORTH and R. CHRISTIE, M. J. Australia **1**:176 (Feb. 9) 1946.

Penicillin-resistant staphylococci developed in vivo were as highly pathogenic for laboratory animals as the sensitive parent organisms. Resistant variants developed in vitro were not pathogenic for laboratory animals. Staphylococci in which resistance had been developed in the human body following penicillin treatment killed mice in spite of the administration of large amounts of penicillin.

GONCE, Madison, Wis.

Metabolism; Infant Feeding; Milk and Other Foods

NUTRITIONAL STUDIES ON MILK FAT: 1. THE GROWTH OF YOUNG RATS FED MILK FAT AND CERTAIN SYNTHETIC GLYCERIDES AS SUPPLEMENTS TO A FAT-FREE DIET. J. L. HENDERSON, E. L. JACK, SAMUEL LEPKOVSKY and DELLA F. REID, J. Nutrition **30**:169 (Sept.) 1945.

A fat-free diet was described in which all the components were pure compounds with the exception of casein extracted with alcohol and ether. Henderson and his colleagues found that rats fed the milk fat and the triolein diets showed a faster rate of growth than those receiving the other diets. Trilaurin restricted growth.

FREDEEN, Kansas City, Mo.

NUTRITIONAL STUDIES ON MILK FAT: II. THE GROWTH OF YOUNG RATS FED GLYCERIDE FRACTIONS SEPARATED FROM MILK FAT. E. L. JACK, J. L. HENDERSON, DELLA F. REID and SAMUEL LEPKOVSKY, J. Nutrition **30**:175 (Sept.) 1945.

Jack and his colleagues presented the results obtained when glyceride fractions from a natural fat—milk fat—were fed to weanling rats as supplements to a fat-free diet. The experimental data showed the diet containing untreated milk fat to be superior to the others. The remainder of the diets, all of which contained fat which had been dissolved in solvent at some stage in its preparation, were divided essentially into three groups on the basis of their growth-promoting ability: The diet containing the minus 53 C. filtrate was considerably superior to the rest; the diet containing the minus 7 C. filtrate and the fat-free diet were the poorest; the others formed an intermediate group. The fatty acid composition of the fats suggested that either the oleic acid content or the total unsaturation might be a factor contributing to differences in growth.

FREDEEN, Kansas City, Mo.

THE AVAILABILITY OF THE IRON IN HAWAIIAN-GROWN VEGETABLES. CAREY D. MILLER and LUCILLE LOUIS, J. Nutrition **30**:485 (Dec.) 1945.

Carey D. Miller and Lucille Louis report the results of analysis of the iron in fifteen vegetables and two seaweeds grown in Hawaii and the availability of the iron for the regeneration of hemoglobin in anemic rats.

FREDEEN, Kansas City, Mo.

INDUCTION OF NUTRITIONAL DEFICIENCY BY ORAL ADMINISTRATION OF STREPTOMYCIN. GLADYS A. EMERSON and DOROTHY G. SMITH, J. Pharmacol. & Exper. Therap. **85**:336 (Dec.) 1945.

White rats fed 160,000 to 375,000 units of streptomycin per kilogram of body weight a day grew almost normally for four weeks; after six weeks they became

hyperexcitable and vicious. Rats given 580,000 to 875,000 units were distinctly retarded in growth after the first four weeks, and the toxic effects were greater. Biotin relieved the loss of hair. The intestinal coliform depression was greater than with succinylsulfathiazole and the total flora was more depressed than with that drug, suggesting that temporary sterilization of the gastrointestinal tract might be more effective with streptomycin than with succinylsulfathiazole.

PILCHER, Cleveland.

METABOLIC COMA IN NUTRITIONAL DISTURBANCES IN INFANTS. J. POU DIAZ, Rev. españ. de pediat. **1**:631 (Sept.-Oct.) 1945.

The author proposes in this article the term "metabolic coma" for the severe nutritional disturbances which have been termed "anhydremia," "alimentary intoxication," "cholera infantum," etc. The lowering of the level of sodium is the major cause of the clinical and chemical picture. The author separates these cases into two categories, primary metabolic coma and secondary metabolic coma. The first type appears without significant loss of water, usually as the result of infection which, by adrenal depression as well as by the initiation of vomiting and diarrhea, leads to a deficiency of sodium. This in turn leads to the dehydration and circulatory collapse associated with the condition. Of the secondary type are cases in which there are vomiting and diarrhea preceding the collapse. In these cases, also, the deficiency of sodium is primarily responsible. In vomiting, the kidneys excrete sodium to compensate for an alkalosis, and in diarrhea the sodium lost is in the stool. The author concludes that a loss of sodium is the point of departure in this condition whatever the cause. He also feels that adrenal insufficiency is a factor in the causal mechanism.

MILLER, Newark, Ohio.

CLINICAL REPORT ON DISTURBANCE IN CALCIUM METABOLISM: RECURRENT TETANY WITH CALCIUM DISTURBANCES IN THE BONES. G. FANCONI, Helvet. pædiat. acta **1**:85 (Aug.) 1945.

Hypocalcemic hyperphosphatemic tetany was found in a constitutionally deficient child of 6 weeks with an inborn lability of the calcium metabolism. At 5 months the same child showed tetany due to deficient vitamin D. Roentgenograms made when the patient was 6 weeks old showed periosteal elevation of the shafts of the long bones probably due to the disturbance of the calcium metabolism and not to lack of vitamin D. The present knowledge of the workings of calcium metabolism and its disturbances is reviewed.

AUTHOR'S SUMMARY.

CYSTINE DISEASE (AMINE DIABETES): FURTHER REPORTS. G. FANCONI, Helvet. pædiat. acta **1**:183 (Feb.) 1946.

Three cases of cystine disease are described. Two of these (cases 2 and 3) could be studies of the condition preceding renal insufficiency. Inheritance is probably simple recessive. Disturbance of development does not begin until several months after birth. Dwarfism, anorexia, obstipation, polyuria, permanent albuminuria, intermittent glycosuria, especially in the initial stage, thermolability and inclination to intoxication—even ingestion of sugar produces a hyperglycemic shock—and hypoplasia of the thymus gland are characteristic of the disease. The bones first show signs of osteodysplasia (pseudorachitis) made evident by osteoporosis, cupping of the metaphysis and incurvation of the normally calcified epiphysial line. This osteodysplasia is followed by true rickets healed by vitamin D though only slowly (early stage) as well as by hyperphosphatemic renal rickets (late stage). In the regularly alkaline urine the organic acids, the amino acids (including especially cystine except in the terminal stage) and ammonia are greatly increased. In spite of the alkaline urine there exists regularly an intermediary acidosis (low alkali reserve, tendency to hypochloremia, increase of the serum protein. etc.).

The method of choice for rapid diagnosis intra vitam of the storage of cystine consists in finding the cystine crystals in the bone marrow obtained by puncture.

Probably not only the decomposition of cystine is disturbed but the oxidative deamidization in general so that besides the cystine other amino acids and even amines (causing the alkaline reaction of the urine) are secreted or retained. The only slightly soluble cystine is stored visibly in the reticuloendothelial system. Perhaps the name "diabetes acidaminicus et aminicus"—or "amine diabetes"—would be better and more accurate.

By a diet rich in base equivalents and vitamin D and by administration of sodium bicarbonate and eventually yeast or the entire vitamin B complex, the condition may be improved and life prolonged for a short time.

AUTHOR'S SUMMARY.

PATHOLOGIC ANATOMY OF CYSTINE DISEASE AS REVEALED IN A CHILD WHO DIED IN AN EARY STAGE. PETER WASER, Helvet. pædiat. acta **1**:206 (Feb.) 1946.

The early case described (case 3 in Fanconi, G.: Cystine Disease [Amine Diabetes]: Further Reports, *Helvet, pædiat. acta* **1**:183 [Feb.] 1946) showed cystine accumulation of a medium degree in the reticuloendothelial system, especially in the liver, the spleen, the lymph nodes and the bone marrow. Liver, spleen and numerous lymph nodes were enlarged. Especially in the liver cystine-containing granulomas consisting of nodelike proliferated Kupffer cells and a few inflammatory cells were found. The renal lesions were diffuse glomerulonephrosis, advanced tubulonephrosis and interstitial nephritis with a small amount of cystine embedded in the interstitial tissues. The kidneys were of normal size. The skeleton was that of a well proportioned dwarf, and a few epiphysial remains of a healing rachitis were found. Formation of osteoid tissue, dissecting resorption and especially osteoclasis and extensive fibrosis of the bone marrow, as observed in cases of chronic renal insufficiency, were missing.

AUTHOR'S SUMMARY.

Vitamins; Avitaminoses

RICKETS IN ICELAND. NIELS DUNGAL, Am. J. M. Sc. **210**:70 (July) 1945.

A survey was made of 253 children in Iceland, all between 3 months and 2 years of age. A clinical and roentgenologic examination was made of each child's wrist and knee, and usually also of his crus and ankle.

The clinical examination showed definite signs of rickets in 66 per cent. Of these, 35 per cent had a visible Harrison's groove, 44 per cent a rachitic rosary, and definite signs of cranial rickets were found in at least 54 per cent. Roentgenographs showed signs of rickets in 75 per cent of the children.

The final result, obtained by comparison of clinical and roentgenologic data, showed that 77 per cent had rickets.

These results dismiss the widespread belief that rickets is unknown in Iceland.

HENSKE, Omaha.

SUPPRESSION OF CIRCULATING ANTIBODIES IN PYRIDOXIN DEFICIENCY. HERBERT C. STOERK and HERMAN N. EISEN, Proc. Soc. Exp. Biol. & Med. **62**:88 (May) 1946.

In a study of 24 albino rats in three groups: (1) rats fed a pyridoxin-deficient diet, (2) those given restricted amounts of a complete diet so as to duplicate the retardation of growth in the first group and (3) those fed a complete diet, it was found that the weight of the thymus gland was far less in the pyridoxin-deficient animals The animals were immunized against washed sheep cells, and the circulating antibodies were also less in the pyridoxin-deficient rats.

HANSEN, Galveston, Texas.

Hygiene; Growth and Nutrition; Public Health

CALCULATION OF NORMAL WEIGHT. MAURY MASSLER, Child Development **16**:111 (March-June) 1945.

If the human body is regarded as an irregular cylinder, its weight can be expressed as: $\frac{(\text{Mean Circumference})^2}{K} \times \text{Height} \times \text{Specific Gravity}$. The mean circumference of the body was found to be best expressed at the level of the xiphoid process after quiet expiration for both male and female at all ages from birth to old age. Specific gravity taken after quiet expiration revolves about 1.0 as a mean but varies slightly at different ages, in different types of body and in males and females. The formula was tested in more than 1,000 healthy subjects ranging in age from birth to 50 years and finally derived as :

$$\frac{(\text{Circumference of Chest at Xiphoid})^2}{4.0} \times \text{Height} \times 1.0 = \text{Calculated Weight}$$

The predictive value of the formula was found to be good in the majority of cases. The coefficient of correlation between calculated and observed weight was 0.82. Measurement is not difficult in technic but must be done carefully. The technic is described and two nomograms are appended to facilitate calculation.

AUTHOR'S SUMMARY.

CHILD WELFARE WORK IN BRAZIL. E. H. CHRISTOPHERSON, J. Pediat. **28**:327 (March) 1946.

Brazil has advanced considerably in medical education. About two fifths of the physicians south of the United States are located in Brazil, which contains forty-five million persons, or one third of the population of Latin America.

Preventive medical work in Brazil is directed by the National Health Department. Under this department are several special services, many of which have done outstanding work in the control of yellow fever, malaria, leprosy and tuberculosis. There is a National Children's Department, which is primarily a stimulating agency. Its budget is small and its possibilities limited at present.

Throughout the country, each state maintains a health department. It is these state health departments that do the major part of the health and preventive medical work. Their efforts have been directed primarily at the population centers, so that the larger cities have fairly good health facilities, but the smaller towns and rural areas have practically none.

There are a fairly large number of hospitals in Brazil, some one thousand three hundred in all, containing ninety-five thousand beds. Hospital service is not nearly adequate and is limited because of the lack of nursing facilities. There are only about six hundred graduate nurses in the country.

The chief health problems in Brazil are the basic ones of sanitation and education. The greatest cause of illness and death is acute intestinal disease, which accounts for 350 deaths per hundred thousand persons. Tuberculosis causes 300 deaths and pneumonia and influenza 200.

The greatest cause of infant deaths is also dysentery, followed by respiratory infections and tuberculosis. The infant mortality rate is 185 per thousand live births. This situation has not changed for thirty years. In communities with not so well developed health facilities the reported infant deaths run as high as 450 per thousand live births.

There are many other prevalent diseases that may not cause many deaths, but they are extremely debilitating and contribute to much mortality and incapacitate great numbers. Chief among these are intestinal parasites, particularly hookworm and amebiasis, malaria and malnutrition.

It will be impossible materially to affect the morbidity and mortality rates of the prevalent diseases in Brazil without a great program of health education and a basic health program. To control these diseases, which are caused by sanitation and environmental problems, a great community interest must be created.

The Special Health Service organized by the Institute of Inter-American Affairs and the Ministry of Education and Health of Brazil developed a health program in Brazil in an attempt to meet these problems.

There can be no successful child welfare work in Brazil until the basic health work is done, such as health education and the providing of good environmental sanitation.

AUTHOR'S ABSTRACT.

GROWTH AND HEALTH OF BELGIAN CHILDREN. RICHARD W. B. ELLIS, Arch. Dis. Childhood **20**:97 (Sept.) 1945.

The general health of Belgian children during the war is discussed. With the exception of rickets, deficiency diseases do not appear to have been of exceptionally high incidence, but there is thought to have been a considerable increase in osteomyelitis associated with sepsis of the skin.

A detailed study of a working class urban family with three children is given, and the weekly budget in February 1945 discussed.

Height and weight curves of children living in a working class urban commune are presented for 1942, 1943 and 1944. While there has been a steady and substantial improvement since 1942, there is evidence that the average values in the autumn of 1944 were still substantially below those for 1939. This was clearly seen in boys in employment aged 14 to 16.

Adolescents appear to have suffered more than the groups of younger age, and a study of 106 adolescent girls indicates a retardation of puberty in a significant proportion of those living at home in a working class commune.

It is considered that in the winter of 1944-1945 Belgian children as a whole were suffering more from lack of fuel, warm clothes and soap than from lack of calories, although the food situation was still unsatisfactory.

KELLY, Milwaukee.

Prematurity and Congenital Deformities

FETAL ELECTROCARDIOGRAPHY. P. VARA and E. HALMINEN, Acta obst. et gynec. Scandinav. **26**:249, 1946.

The authors briefly review the literature dealing with fetal electrocardiography. They present the results in 30 cases of their own, in which fetal electrocardiograms were obtained by the use of a standard electrocardiographic machine with appropriate amplifier. Maternal leads were taken from the abdomen and either the suprapubic area or the anterior vaginal vault. Tracings from 8 of the patients are reproduced. The fetal heart waves are found to be superimposed on the maternal electrocardiogram, the latter being considerably distorted by the excessive speed at which it was necessary to move the drum. The method is presented as having practical value in the diagnosis of fetal death and, since records are said to be obtainable as early as three months, in the diagnosis of early pregnancy. The fetal waves indicate only whether the heart is beating, and provide no information as to the cardiac status of the fetus.

DANFORTH, Chicago.

Newborn

DIAGNOSIS OF ERYTHROBLASTOSIS (HEMOLYTIC ANEMIA) IN THE MACERATED FETUS. EDITH L. POTTER, Arch. Path. **41**:223 (March) 1946.

Dr. Potter refers to her previous observations that in a series of 2,000 infants dying in utero or in the neonatal period, 19 per cent were infants dying before the onset of labor and in whom no cause of death could be determined. She stresses the importance of definite criteria for the diagnosis of erythroblastosis in these macerated infants, and warns against advising Rh-negative mothers to forego future pregnancies unless definite evidence of the disease is found in the fetus. Her criteria for the diagnosis of erythroblastosis in macerated premature or term

fetuses include: edema, especially of the face; mild macroglossia; hepatomegaly; splenomegaly; normal ossification lines in the long bones; enlargement of the placenta; erythroblasts in the placental and fetal pulmonary vessels; absence of Rh in the mother's red cells and presence of Rh in the father's red cells. The chief differential diagnoses considered are syphilis and extreme hydrosis (a condition previously described by the author). She regards the lungs as the most valuable organs of the macerated fetus in this disease. They retain their staining ability longer than the other organs, and the presence of large numbers of immature red cell forms in the alveolar capillaries has been found in her experience only in the fetus and the newborn infant with erythroblastosis. The placental changes described include: increased weight, well demarcated cotyledons, enlarged and edematous villi and immature red blood cells in the vessels of the major villi.

GEREN, St. Louis.

RESUSCITATION OF THE ASPHYXIATED BABY DELIVERED BY CESAREAN SECTION. J. D RUSS, New Orleans M. & S. J. **98**:415 (March) 1946.

The popular belief in the safety of the infant delivered by cesarean section is not supported by facts. When all technics of delivery are considered, the standard neonatal mortality is 2.5 to 3 per cent. In cesarean sections in general hospitals the rate is 8 to 10 per cent and in the best private hospitals 5 to 6 per cent. Deaths are attributed to asphyxiation, aspiration pneumonia (atelectasis) and cerebral hemorrhage. Asphyxia and cyanosis are outstanding symptoms in all three.

The author believes that some one versed in the art of resuscitation should be present at all cesarean deliveries. The following procedures are considered essential: 1. When the baby has been delivered, allow the cord to pulsate for a few minutes and then strip it toward the baby before severing it. 2. Receive the baby into a warm blanket, place it on a flat table, and with an intratracheal catheter aspirate the nose, the mouth, under the gums, the cheeks and the posterior part of the nasopharynx. 3. Then enter the trachea and aspirate gently the trachea and the upper respiratory tract. 4. This accomplished, then by means of the resuscitator's own breath the baby's lungs should be inflated. This should be repeated twenty-four to thirty times per minute until the baby has taken one or two spontaneous breaths. 5. Remove the tracheal catheter and give pure oxygen by face mask. Continue for two to five minutes until the breathing is rhythmic, the color is a healthy pink and the infant shows a tendency to cry or has begun to cry.

Use of this method in 317 cases resulted in only 6 neonatal deaths before the end of the first twenty-four hours. In 5 of the 6 fatal cases autopsies were done, and massive cerebral hemorrhages were found.

In 137 sections in which no routine intratracheal catheterization was performed there were 12 deaths. In 11 cases autopsies were done; 2 infants were found to have died from cerebral hemorrhage and 9 from aspiration pneumonia. The death rate in the first series was 1.8 per cent and in the second 8.7 per cent. The results substantially support the author's contentions and methods.

BERKLEY, Beverly Hills, Calif.

RUPTURE OF THE LIVER IN THE NEW-BORN: RECOVERY AFTER BLOOD TRANSFUSION AND LAPAROTOMY. F. ARDEN, M.J. Australia **1**:187 (Feb. 9) 1946.

Thirty-six hours after a relatively normal delivery this infant, whose birth weight was 9 pounds (4,082 Gm.), showed pallor, breathlessness and inability to suck, and over the next two hours an increase in pallor, air hunger, an almost imperceptible pulse and abdominal distention developed, with dulness to percussion all over the right side and in the left flank. A transfusion of 200 cc. of blood was given rapidly and followed by another 100 cc. over the next four hours. Operation was then performed (when the infant was 42 hours old); the peritoneal cavity was found to be distended with blood and blood clot, and the liver was found to be

torn by a fissure 2 inches (5.1 cm.) in length situated on the anterosuperior surface of the right lobe close to the midline. The torn edges of the liver capsule were sutured to the cut edges of the parietal peritoneum, a strip of right rectus abdominis muscle, with a pedicle at the lower end, placed in the hepatic substance, the abdominal wall closed and firm pressure applied by strapping over the dressings. Recovery was uneventful and at the age of 7 months the infant "was in magnificent condition and weighed 20 pounds [9,072 Gm.]."

GONCE, Madison, Wis.

RENAL VENOUS THROMBOSIS AND INFARCTION IN THE NEWBORN. J. EDGAR MORISON, Arch. Dis. Childhood **20**:129 (Sept.) 1945.

In 5 newborn infants thrombi formed in the intrarenal veins without producing infarction of the kidney. In another 5 cases thrombi resulted in hemorrhage between the tubules of some renal papillae; in 2 others there was extensive disruption, especially of the medulla, by effused blood, and 2 cases showed both red and pale venous infarcts. These cases are compared with 4 in which there was hemorrhagic infarction without thrombi in the veins and with 4, 2 of which were within the neonatal period, in which similar but less pronounced lesions resulted from pyelonephritis.

Thrombi were organized or covered by endothelium in 11 cases and had formed first in the small intrarenal branches. Dehydration, continued sepsis and renal venous stasis, but not renal inflammation, appeared to be important in their causation. Emboli in the small pulmonary arteries and sometimes small areas of infarction occurred in the lungs in 9 of the 14 cases of idiopathic renal venous thrombosis.

KELLY, Milwaukee.

ENDOUTERINE TRAUMATIC FRACTURE OF THE RIGHT CLAVICLE WITH CONGENITAL PARALYSIS OF AN ISOLATED BRANCH OF THE CERVICOBRACHIAL PLEXUS. E. FORLINI, Riv. di clin. pediat. **44**:85 (Feb.) 1946.

A rare endouterine traumatic fracture of the right clavicle in a newborn girl, caused by the mother's falling and striking the lower part of her abdomen twenty-five days before delivery, is described. A few days after birth a well formed callus of the right collarbone and a congenital paralysis of an isolated branch of the cervicobrachial plexus, innervating the "dentatus maximus," was noted.

For this reason the interior margin of the scapula was close to the spinal column because of the antagonistic action of the rhomboid muscles.

No defects in ossification or calcification due to "osteogenesis imperfecta" or syphilis were visible in the roentgenograms of the other bones.

The Wassermann reaction of the parents was negative.

The collarbone was fractured in the third external portion, in the middle of the posterior convexity, as in the case of indirect fractures.

The external portion was deflected upward, and the internal portion downward; both were contained in the callus.

Some months after birth the superior margin of the collarbone showed almost a continuous surface and the paralysis had disappeared.

Clinical aspects of the case are considered.

AUTHOR'S SUMMARY.

Acute Contagious Diseases

A STUDY OF THE PREVENTION OF MUMPS ORCHITIS BY GAMMA GLOBULIN. SYDNEY S. GELLIS, AIMS C. MCGUINNESS and MICHAEL PETERS, Am. J. M. Sc. **210**: 661 (Nov.) 1945.

The authors demonstrated that gamma globulin derived from pools of normal human plasma is ineffective in the dosage employed at present in reducing the incidence of orchitis complicating mumps, whereas gamma globulin from convalescent serum appears highly effective in this respect and warrants further investigation.

HENSKE, Omaha.

A Note on the Use of Penicillin in the Laboratory Diagnosis of Whooping-Cough. Phyllis M. Anderson, M. J. Australia **1**:244 (Feb. 23) 1946.

The author found the incorporation of penicillin into the Bordet-Gengou medium to greatly facilitate the bacteriologic diagnosis of whooping cough. Hemophilus pertussis was isolated from 79 to 154 patients by means of the penicillin plate method and from only 39 of them when ordinary medium was used.

Gonce, Madison, Wis.

Heine-Medin Disease in the Province of Modena in the Quadrennium Following the Epidemic of 1939: Epidemiologic Report and Immunologic Consideration. F. Fontana, Riv. di clin. pediat. **44**:65 (Feb.) 1946.

Eighty-one cases of Heine-Medin disease which occurred in the Province of Modena during the four years following the 1939 epidemic are described. The author considers the topographic distribution and its relation with the different epidemiologic factors.

From the immunologic point of view, the investigation on the age and residence of children affected by Heine-Medin disease in the postepidemic period permits some interesting considerations, especially on the conception of the "silent immunization."

Author's Summary.

Malignant Diphtheria and Pathogenic Importance of Bacterial Mesomucinases. A. Galeotti Flori, Riv. di clin. pediat. **44**:92 (Feb.) 1946.

On the grounds of the recent reports on the bacterial mucinolytic enzymes (mesomucinases) and of the research work to which the author contributed experimentally, the clinical elements and the evidence of the pathogenetic importance of the bacterial mucinolytic enzymes in malignant diphtheria are expounded.

Author's Summary.

Some Clinical Considerations in a Case of Heine-Medin Disease in One of Monochorionic Twins. E. Messeri, Riv. di clin. pediat. **44**:100 (Feb.) 1946

A description is given of an unusual case of poliomyelitis in 2 monochorionic twins in whom the onset of the illness was simultaneous and with mirror symmetry. The pathogenesis of this disease is discussed on genetic and embryologic grounds.

Author's Summary.

Immunization Methods by Means of Diphtheria and Tetanus Anatoxin and the Prophylaxis of Diphtheria and Tetanus. G. Ramon, Bull. Schweiz. Akad. d. med. Wissenschaften **1**:413, 1946.

The method of vaccination against diphtheria based on the use of diphtherial anatoxin (toxoid) is actually the most in use. Compulsory in certain countries (several Swiss cantons, Hungary, Poland, Rumania, France U.S.S.R., Norway, Argentina, Spain, etc.), it is practiced systematically in most of the others: England, Canada, Denmark, the United States and others. Results obtained since 1923, and especially those in recent years in Switzerland, as well as in France, Great Britain, the United States and other countries, incontestably confirm the efficacy of vaccination with diphtherial anatoxin. The conviction that diphtheria can be made to disappear is becoming more and more likely by the generalized application of this method of specific prophylaxis, which, wherever it has been correctly and judiciously used, in a large enough number of persons, has resulted in a considerable reduction in the morbidity and mortality caused by diphtheria.

The information collected during the World War in the armies of America, England, France and other countries, has given clear and indisputable proof of the

efficacy of vaccination against tetanus by means of tetanic anatoxin and this under the most rigorous conditions, those of war, which greatly increase its demonstrative value.

New procedure of specific treatment of toxic infections such as diphtherial serum anatoxin therapy and tetanic serum anatoxin therapy, which are based on the simultaneous use of specific antitoxins and anatoxins, have also been put to the test during the last few years and have not been found wanting.

All these methods have but one objective, which coincides with that of the International Red Cross: to relieve human suffering and to cheat death (Pasteur).

AUTHOR'S SUMMARY.

Acute Infectious Diseases

THE ERYTHROCYTE SEDIMENTATION RATE IN RHEUMATIC FEVER: ITS SIGNIFICANCE IN ADOLESCENT AND OVERWEIGHT CHILDREN. T. N. HARRIS, Am. J. M. Sc. **210**:173 (Aug.) 1945.

Four hundred children, 215 of whom were girls, were observed during their convalescence from episodes of rheumatic carditis. In 9 of these patients, 8 girls and 1 boy, ranging from 9 to 16 years of age, the erythrocyte sedimentation rate remained persistently elevated long after the other signs, symptoms and tests for active rheumatic infection had returned to normal or become stationary. These patients were ultimately allowed out of bed while the erythrocyte sedimentation rate was still elevated. Continued clinical observation following the resumption of physical activity failed to reveal any indication that these children had been allowed out of bed prematurely.

Some theoretic and clinical implications of these data are discussed.

HENSKE, Omaha.

PENICILLIN THERAPY OF STAPHYLOCOCCUS AUREUS HEMOLYTICUS INFECTION IN AN ALLERGIC INFANT. GOLDA FISCHER and LAURA E. MCCLURE, J. Pediat. **27**:436 (Nov.) 1945.

This is a report of a case of a 5 month old Negro boy with severe allergic tendencies, complicated by infection. During his one hundred and twenty-seven days of hospitalization he had clinical evidence of persistent infection of the upper respiratory tract, bronchitis and aphthous stomatitis, three episodes of diarrhea, furunculosis of the scalp and the forehead with cervical lymphadenopathy and multiple subcutaneous abscesses. Throughout his stay he had a septic type of elevation of temperature, the rectal temperature frequently exceeding 104 F.

Aside from frequent increase in leukocyte count, the only abnormal laboratory findings were cultures of Staphylococcus aureus hemolyticus from the furuncles of the scalp and the subcutaneous abscesses and a predominance of Staphylococcus aureus in the throat culture. The possible exception is the report of Staphylococcus anhemolyticus in the subculture of the first blood culture. This was interpreted as a contamination at the time, as the child was clinically improved.

Maintenance of nutrition and of fluid intake was always difficult because of the severe allergy. Intravenous therapy was made difficult because of the weeping eczematous areas in the antecubital and cervical areas and the crusted lesions about the wrists and the ankles. On this account parenteral solutions were usually given by hypodermoclysis.

On the fifty-eighth day furunculosis of the scalp developed. Culture from one of the furuncles showed Staphylococcus aureus hemolyticus. On the sixtieth day penicillin therapy was begun, 5,000 units being administered every two hours up to a total of 200,000 units in four days. As the local lesions had cleared and penicillin was difficult to obtain at the time, its administration was discontinued even though some elevation in temperature persisted.

Two weeks later the child became critically ill, with rectal temperature elevated to 106 F., abdominal distention, vomiting and watery stools containing mucus, pus and blood. Fluids were given subcutaneously, including a solution of amino acids. Administration of penicillin was resumed in doses of 5,000 units every two hours. Indurated areas appeared at the site of each hypodermoclysis. One area was incised, and pus was obtained. A second was aspirated, and a culture was made from the pus. This showed a pure culture of Staphylococcus aureus hemolyticus. An autogenous vaccine was prepared. Penicillin was increased to 10,000 units every two hours, or 120,000 units daily. After six days it was reduced to 60,000 units daily. A total of 2,000,000 units of penicillin was given. All abscesses cleared, and the child became clinically well. Administration of the autogenous vaccine was continued in the outpatient department.

The authors expressed the opinion that this was a staphylococcic septicemia with metastatic abscesses at the site of tissue trauma due to the hypodermoclysis. It is of interest because of the large quantity of penicillin required to effect a cure.

AUTHORS' ABSTRACT.

PENICILLIN THERAPY IN RATBITE FEVER. FRED BROOKSALER, J. Pediat. **27**:442 (Nov.) 1945.

The two separate forms of rat-bite fever are briefly reviewed. A case of Streptobacillus moniliformis septicemia following rat-bite in an 18 month old child is presented with clinical and laboratory observations. This patient showed a prompt and permanent therapeutic response to penicillin. In vitro the strain of Streptobacillus moniliformis recovered was definitely susceptible to the bacteriostatic effect of penicillin. Penicillin is recommended as the treatment of choice in rat-bite and in Haverhill fever.

AUTHOR'S SUMMARY.

UNUSUAL FULMINATING GENERALIZED STAPHYLOCOCCUS INFECTIONS TREATED WITH PENICILLIN. LEO BASHINSKY, J. Pediat. **27**:448 (Nov.) 1945.

The author reports on 2 cases of generalized staphylococcic infections. In the first case a 2 week old boy with staphylococcic septicemia had abscesses over the liver which communicated with that organ. After four weeks of penicillin, the recovery appeared complete. In the second case in a 6 year old boy with congenital ptosis of the right eyelid exophthalmos and stiff neck developed. Cultures revealed hemolytic staphylococci from the stool, the urine, the blood, the spinal fluid and the nose. The final diagnosis was hemolytic Staphylococcus aureus cavernous sinus thrombosis, septicemia, meningitis, pyelitis and enteritis. With the patient under treatment with penicillin, the temperature dropped in five days, and the child by the tenth day was subjectively well.

HISTOPLASMOSIS. JOHN H. MCLEOD, CHESTER W. EMMONS, SIDNEY ROSS and FREDERIC G. BURKE, J. Pediat. **28**:275 (March) 1946.

Histoplasmosis was first observed and described by Darling in 1906, and he reported 3 cases in adults. Observation of additional cases showed that the disease was not confined to the tropics and that it was not confined to adults. In 1934 Dodd and Thompson reported a case of this mycosis in a 6 month old child living in Tennessee. In the excellent summary review of the disease made by Parsons and Zarafonetis, they pointed out that in 11 of 60 cases for which age data were available the patients were less than 1 year of age. Histoplasmosis is of special interest to pediatricians not only because of its apparent predilection for infancy but also because of the suggestion made by a number of investigators that this mycosis, fatal in nearly all recognized cases, may occur in a benign form which is now unrecognized and that therefore it may be much commoner in childhood than is generally supposed.

This report represents a clinical summary of 4 cases of histoplasmosis seen at Children's Hospital, Washington, D. C. Two of the cases, hitherto unreported,

occurred in brothers and represent the first reported instances of histoplasmosis in siblings. These 4 cases are presented, not only because they extend further the wide range of clinical variation of the disease already known, but also because they raise important epidemiologic considerations. All 4 patients were children living in Loudoun County, Va., within a radius of 8 miles (12.9 kilometers) of Leesburg, suggesting that there may be in this area an unusual geographic concentration of the mycosis. In addition the occurrence of histoplasmosis in siblings suggests that there may have been transmission from one person to another.

There is considerable variation in the symptoms and the physical observations in histoplasmosis. Irregular temperature is an almost constant feature of the disease. Anemia, hepatomegaly, splenomegaly and lymphadenopathy are present in the majority of cases. Other manifestations of the disease include leukopenia, gastrointestinal symptoms, emaciation and ulcerations of the mouth and buccal cavity. The disease may simulate aleukemic leukemia, Hodgkin's disease, lymphosarcoma, tuberculous adenitis, kala-azar, brucellosis, malaria, infectious mononucleosis and neoplasm.

The diagnosis of histoplasmosis can be established only by the laboratory demonstration of the fungus. Biopsy of the lymph nodes, spleen and material from ulcerations probably constitutes the most reliable method of making the diagnosis of histoplasmosis, although the organisms have been demonstrated on peripheral blood smears, blood cultures and aspirated bone marrow.

The cutaneous test with histoplasmin is not specific for histoplasmosis but shows cross reactions with blastomycosis, coccidiomycosis, haplomycosis and Candida albicans.

The portal of entry of Histoplasma capsulatum is not known. Parasitic infections, infected bites, ingestion and inhalation have all been suggested as possible modes of entry, with inhalation and ingestion the more likely possibilities. In the instance of its occurrence in siblings noted in this report, it is impossible at this time to determine whether the disease was transmitted from one of these brothers to the other. Obviously, the opportunity for transmission was good because the boys slept together at a time when one of them had gastrointestinal symptoms. Transmission is theoretically possible, but if it commonly occurs, it is not recognized.

The course of the disease is characterized by its terminal progression; it may last from weeks to years. The disease may have an acute onset and terminate rapidly, while, on the other hand, it may appear in a chronic form with irregular exacerbations and long remissions.

There is no specific therapy for histoplasmosis. Various drugs including sulfonamide compounds, quinine, ammonium and potassium tartrate, arsenicals, pentnucleotide, iodides and others have been used without any singular success. Neostam (nitrogen glucoside of sodium paraaminophenylstibonate) has been used in several cases with equivocal results. The occasional encouraging outcome with this drug would indicate that it warrants further trial. Roentgen therapy does not seem to alter the course of this disease.

AUTHORS' ABSTRACT.

RHEUMATIC RECRUDESCENCES: DIAGNOSIS AND PREVENTION. ARILD E. HANSEN, J. Pediat. **28**:296 (March) 1946.

One of the most characteristic features of rheumatic fever is its propensity to occur in repeated attacks, and it is these recurrences or recrudescences which cause the high death rate and high incidence of disability from damaged heart in children and young adults.

Less than 1 per cent of those afflicted die during the first attack; yet long term follow-up studies disclose that within ten to twenty years 25 to 40 per cent have succumbed to the diseases mostly because of the effects of the repeated attacks. Military experience in World War II revealed both high incidence of disability due to previous rheumatic heart disease and the occurrence of a surprising number of acute attacks of the disease, which in most instances represented recrudescences of previous rheumatic afflictions. Hence, one of the chief problems of rheumatic

fever concerns control of the recurrences. In this presentation rheumatic recrudescences are discussed from the viewpoint of (1) general features, (2) manifestations, (3) diagnosis and (4) methods of prevention.

From 66 to 85 per cent of children with rheumatic fever have one or more recurrences. The younger a child is at onset of the disease the longer is the period in which recurrences may develop; however, recurrences before and after the age of 16 years are in the ratio of 5 to 1. Fully four fifths of the recrudescences develop within five years after an attack of rheumatic fever, but experience in the military forces during the war indicate that often many years may intervene between the first attack and a recurrence. Recurrences may manifest themselves in a variety of ways. In about 75 per cent of the children with acute rheumatic fever the onset is characterized by (1) polyarthritis, (2) mild articular pains or (3) rheumatic encephalitis (chorea) equally distributed. However, in the course of years 66 per cent will have attacks of severe polyarthritis, the same percentage will have attacks of mild articular pains, and 45 per cent will have chorea. Only 3 per cent will have an onset characterized by severe carditis without other manifestations, and only 1 per cent will have congestive failure during the initial attack. Eventually, 75 to 80 per cent will develop severe carditis, and 25 per cent will have experienced congestive failure.

The great variety of manifestations of rheumatic fever which may be present in a recrudescence often makes differential diagnosis difficult. A series of studies revealed that when pain was a pronounced feature the diseases most apt to be confused in the differential diagnosis of rheumatic fever were: appendicitis, poliomyelitis, osteomyelitis and rheumatoid arthritis.

Much has been accomplished in recent years to prevent recrudescences. Proper convalescent care and supervision are of prime importance, and change in climate may be worthy of consideration. The continuous use of sodium salicylate for a month following a throat infection with group A hemolytic streptococci appears to prevent a rheumatic flare-up from developing. Economic factors preclude the wide use of such a measure. Immunizations with tannic acid–precipitated toxins of hemolytic streptococci seem to offer distinct benefit. The most widespread method of prevention is to administer relatively small amounts of sulfanilamide (0.6 Gm. twice daily) or sulfamerazine or sulfadiazine (0.5 Gm. twice daily) continuously. Smaller doses may be used for the younger children. There should be no signs of activity of the rheumatic infection when such therapy is started, and it is necessary to keep the patient under careful observation by the physician with periodic examination of the blood and the urine when sulfonamide compounds are used prophylactically. Many workers have reported studies comprising an experience of 1,561 patient-seasons, and it has been found that the recurrences were only 1.5 per cent in those patients given sulfonamide compounds in contrast to 19 per cent for control patients who were not given the drug. Further work is necessary to determine how long such therapy should be continued. For children it would appear that such a regimen should be carried on for five years following an attack of the disease, or until the age of 17 years, when recrudescences are less likely to develop. Nonetheless, it should be emphasized that the physician who takes care of the child with rheumatic fever should have him under careful scrutiny at all times; thus much may be done to reduce the high mortality and disability in rheumatic fever by the prevention of recurrences.

AUTHOR'S ABSTRACT.

THE PRESENT STATUS OF THE USE OF TETANUS TOXOID. FRANK G. GRUICH, New Orleans M. & S. J. **98**:420 (March) 1946.

The actual level of antitoxin in the blood required to produce clinical immunity to tetanus has not been definitely established. Many believe that human beings require titers of 0.01 to 0.1 unit of passively introduced antitoxin while an active immunity requires only 0.001 to 0.01 unit.

The Army's method of immunization requires three injections of 1 cc. each (fluid tetanus toxoid) given three weeks apart. A booster dose of 1 cc. is given one year after the initial series.

The Navy gives as a basic immunization two injections of alum-precipitated tetanus toxoid eight weeks apart. The wounded get another dose at the time of injury, and a refresher dose is given all personnel every four years after the basic doses.

Active immunization with tetanus toxoid raises the serum antitoxin titer to a higher level than that afforded by the usual dose of antitoxin (horse serum). The titer is lasting and can be elevated by another injection when the emergency exists. The real danger of reaction or sensitization when using antitoxin is, for the most part, avoided in the use of toxoid.

Up to the present time there have been no reported cases of tetanus following the completion of basic active immunization in the armed forces of the United States.

BERKLEY, Beverly Hills, Calif.

AUTOANTIBODIES IN RHEUMATIC FEVER. PHILIP A. CAVELTI, Proc. Soc. Exper. Biol. & Med. **60**:379 (Dec.) 1945.

Antigens from saline extracts of human hearts were prepared by the collodion particle technic (Cavelti, P. A.: *J. Immunol.* **49**:365, 1944), and appropriate amounts were added to a series of tubes containing progressive dilutions of serum from (1) normal persons, (2) patients with a variety of diseases and (3) patients with rheumatic fever. After incubation for one hour the tubes were centrifuged and the results of agglutination read plus-minus to 4 plus. Antibodies to human heart were demonstrated in the majority of the serums from patients with active rheumatic fever. Twenty of the 36 patients had strong reactions; 7 had weak but positive reactions, and the remaining 9 had doubtful or negative reactions. In periodic studies (two to five weeks) of these patients, 47 specimens of the 67 examined were definitely positive. The serums from 12 control subjects gave completely negative results, and of the serums of 84 patients with various diseases 1 (chronic myeloid leukemia) showed a weakly positive reaction, and 3 others had doubtful reactions.

HANSEN, Galveston, Texas.

EFFECT OF SALICYLATE ON PLASMA FIBRINOGEN AND SEDIMENTATION RATE IN RHEUMATIC AND NON-RHEUMATIC PATIENTS. S. RAPOPORT and GEORGE M. GUEST, Proc. Soc. Exper. Biol. & Med. **61**:43 (Jan.) 1946.

The concentration of fibrinogen in the plasma and the sedimentation rates of erythrocytes were determined before, during and after the administration of acetylsalicylic acid (0.09 to 0.22 Gm. per kilogram of body weight with an equal amount of sodium bicarbonate) for seven to twenty-nine days to 15 patients (8 with some manifestation of rheumatic fever, 4 with lipid nephrosis and 3 suffering from chronic osteomyelitis) from 2 to 14 years of age all of whom had elevated sedimentation rates. In 10 patients the sedimentation rate and the concentration of fibrinogen in the plasma were reduced after the use of salicylates. In the other 5 patients, in whom the concentration of fibrinogen in the plasma did not decrease, no important changes were observed in the sedimentation rates. In several instances there were discrepancies between the two indexes. The authors state that it is suggested that the change in the fibrinogen may represent an effect of salicylate on the liver.

HANSEN, Galveston, Texas.

IMMUNOLOGICAL STUDIES OF DENGUE FEVER AND COLORADO TICK FEVER. M. POLLARD, H. R. LIVESAY, D. J. WILSON and J. C. WOODLAND, Proc. Soc. Exper. Biol. & Med. **61**:396 (April) 1946.

Both dengue fever and Colorado tick fever are due to a filterable agent implicated with an arthropod (mosquitoes and ticks, respectively) and are characterized

by generalized malaise, headache, leukopenia and a diphasic or "saddle-back" temperature curve. Both are self limiting and nonfatal. Many patients with dengue fever have a cutaneous rash. Study with 6 human subjects indicated that there is no immunologic relationship between dengue fever and Colorado tick fever.

HANSEN, Galveston, Texas.

Chronic Infectious Diseases

THE RELATION OF THE DEGREE OF SENSITIVITY TO TUBERCULIN TO THE PERSISTENCE OF SENSITIVITY AND TO PROGNOSIS IN YOUNG CHILDREN. LYDIA B. EDWARDS and JANET B. HARDY, Bull. Johns Hopkins Hosp. **78**:13 (Jan.) 1946.

A study of sensitivity to tuberculin was made in 207 children, all under 2 years of age and all reacting positively before the third birthday. They were divided into three groups: those with positive reactions to not less than 1.0 mg. of old tuberculin (34); those with positive reactions to 0.1 mg., but not less (26), and those with positive reactions to 0.01 mg. of old tuberculin or less (147).

Of those reacting only to 1 mg., 56 per cent lost their hypersensitivity and did not regain it. None of the 34 showed demonstrable parenchymal lesions, calcification or extrapulmonary tuberculosis. Of those reacting to 0.1 mg., 23 per cent lost their sensitivity, none died and 23 per cent had parenchymal involvement, calcification or extrapulmonary tuberculosis. Among those reacting to 0.01 mg. or less there was a 12 per cent death rate, and 88 per cent showed some evidence of tuberculosis.

The degree and the maintenance of sensitivity were directly proportional to the extent of the lesion. The prognosis was bad in proportion to the degree of sensitivity.

LYTTLE, Los Angeles.

USUAL AND UNUSUAL FINDINGS IN TUBERCULOSIS OF INFANCY. EDWIN L. KENDIG JR. and JANET B. HARDY, South. M. J. **39**:282 (April) 1946.

The authors emphasize the need for studying infants for the presence of tuberculosis. A positive reaction to the tuberculin test in a child less than 3 years of age probably indicates active infection. Pleurisy with effusion may be caused by tuberculosis in that age group. Of a group of 207 patients who showed a positive tuberculin reaction before their third birthday, those who reacted to no less than 1 mg. of tuberculin did well, while among those who reacted to 0.01 mg. of tuberculin, 45 per cent showed a parenchymal lesion, 27 per cent had an extrapulmonary lesion and 15 per cent died.

LOGAN, Rochester, Minn.

PROPHYLAXIS OF TUBERCULOSIS IN THE CHILD. P. F. ARMAND-DELILLE, Bull. schweiz. Akad. d. med. Wissensch. **1**:438, 1946.

The author states that hereditary tuberculosis does not exist and that children become infected with the disease. The sequels to infection are serious: high mortality among infected babies and later reinfection of infected children with the danger of the occurrence of prognostically unfavorable disseminated pulmonary tuberculosis. Two methods of prophylaxis which should both be used have proved effective. The first method is Calmette's prophylactic vaccination together with Rosenthal's method of scarification. Trosier observed during a period of six years that tuberculosis occurred four times more often in nonvaccinated, tuberculin-negative medical students than in vaccinated students. The second method is isolation as recommended and practiced by Grancher forty years ago; i. e., the separation of the clinically healthy child from its affected parents until the infection has disappeared. Of isolated children 1 in 1,000 becomes infected, but of non-isolated children about 40 per cent. Older children in danger of becoming infected should be in preventoriums.

AUTHOR'S SUMMARY.

Diseases of the Blood, Heart and Blood Vessels and Spleen

AURICULAR FIBRILLATION IN INFANCY. JOSEPH EDEIKEN and STANLEY J. RUGEL, J. Pediat. **28**:471 (April) 1946.

In 1938 Goldbloom and Segall reported a case of auricular fibrillation in an infant 3 months old and stated that an exhaustive search of the literature had not revealed a report of a single case of auricular fibrillation in the first year of life. The authors report a second case first observed by them in 1943; they state that to the best of their knowledge no other report has appeared in the literature since 1938.

The infant was 2 months of age. The birth history revealed that she had been a blue baby. Except for a pronounced irregularity of the heart, physical examination revealed nothing significant, and on July 9, 1943, when the baby was 3 months old, the electrocardiogram was made. This tracing showed paroxysms of auricular fibrillation with rapid ventricular rate. The paroxysms were short and followed a conducted beat. Digitalis was given in small doses without any effect on the arrhythmia or the rapid ventricular rate. When the child was 5 months old, for the first time a high pitched murmur was heard in the second interspace to the left of the midline, and a roentgenogram of the chest revealed the cardiac shadow to be of the "Dutch shoe" type and showed some enlargement. The dose of digitalis was then increased to 1½ grains (0.1 Gm.) a day, and there was slight improvement until the middle of November, when the child became ill. She died suddenly on Nov. 20, 1943.

AUTHORS' ABSTRACT.

SIDEREMIA IN MOTHER AND FETUS. A. GENTILI and A. LATTANZI, Riv. di clin. pediat. **44**:42, 1946.

The authors have studied the behavior of the iron content of the serum and at the same time that of hemoglobinemia and, in some cases, that of bilirubinemia in the mother and in the fetus.

They have found that the iron content of the mother's serum is sometimes considerably diminished, while that of the fetus is notably increased. The average iron content of the serum of the fetus is 100 per cent greater than the average content of the mother's serum. This higher level of iron in the serum of the fetus is supposed to be due to a more intense metabolism of hemoglobin in the fetus than in adults.

AUTHORS' SUMMARY.

HYPOPLASTIC ANEMIA SUBSEQUENT TO THIRD DEGREE MALNUTRITION. JESÚS ALVAREZ DE LOS COBOS, Bol. méd. d. hosp. inf., México **2**:379 (Jan.-Feb.) 1946.

J. C. H. M., an 18 month old girl, was admitted to the hospital because of diarrhea, vomiting and fever. The feeding was extremely poor. The weight was 5,370 Gm. The blood picture showed hypoplastic and hypochromic anemia. The patient was treated intensively with daily transfusions of 100 cc. for ten days, adequate feeding, pyridoxine, penicillin and vitamin B complex. No iron was administered outside the transfusions and the food. The patient was discharged forty-two days later with the return to normal of the blood picture and the myelocyte count, and with a weight of 10,000 Gm. The author discusses the beneficent action of pyridoxine and penicillin.

MADERO, México, D. F., Mexico.

Diseases of Nose, Throat and Ear

THROMBOPHLEBITIS OF THE LATERAL SINUS. GEORGE OTTE, Rev. de otorrinolaring. **5**:173 (Dec.) 1945.

In evaluating thrombophlebitis, Otte states that this is one of the most frequent complications of cases of suppurative otitis media. He believes that its incidence is not influenced by any trauma of mastoidectomy, an opinion contrary to certain

views advanced by other authors. In a group of 100 cases in which the lateral sinus was exposed during operation, he has not in a single case observed subsequent thrombophlebitis, provided frank injury of the vessel had not occurred or the vessel was not previously infected. The symptom complex is usually that of high temperature, with daily remissions, and chills. On the other hand, the patient presents a good general state of health; headaches, mental confusion and other intracranial signs usually are absent, provided there is no associated labyrinthine or meningeal involvement. If intracranial symptoms appear, and one suspects thrombophlebitis, they are an indication of further intracranial complications, most commonly cerebral abscess.

The author discusses the relative merits of ligating the internal jugular vein in a surgical procedure. Personally he prefers ligation, but he believes that in the future, owing to the use of penicillin and other chemotherapeutic agents, this form of surgical intervention may be unnecessary. He presents 2 patients who had a typical form of thrombophlebitis and who were treated intensively with sulfonamide compounds without improvement, for whom it was necessary to perform ligation of the internal jugular vein. One of the side effects of ligation of this vein is intracranial venous stasis, which occurred in one of his patients, with typical hemiplegia, headache, vomiting and facial paralysis. A diagnosis of cerebral abscess was made, and an exploratory puncture was performed both in the cerebrum and in the cerebellum, but the abscess was not found. Subsequent to this, however, the patient made an uneventful spontaneous recovery. Otte presents 3 other patients who had a typical form of thrombophlebitis. Two of them had associated cerebral abscess, and in these the clinical manifestations of thrombophlebitis were definitely obscured by the intracranial symptoms; in the third, ligation of the internal jugular vein was performed, and massive doses of penicillin were administered, but the meningeal symptoms continued, and the patient died. Autopsy revealed tuberculous meningitis. Otte believes that thrombophlebitis is more frequently associated with mastoiditis of the left side and that in consequence the further complication of a cerebellar abscess of that side is more common. On the other hand, if the right mastoid process is involved, there is a greater frequency of cerebral abscess. Therapeutically he believes that the following routine should be enforced: 1. There must be thorough exenteration of the mastoid cells with all diseased tissue and wide exposure of the infected sinus. 2. This is to be supplemented with intensive penicillin therapy. 3. If a septic picture persists, ligation of the jugular vein is indicated. If the bulb of the jugular vein is thrombosed, he advises that it be resected according to the Ramadier technic.

PERSKY, Philadelphia. [ARCH. OTOLARYNG.]

CEREBELLAR ABSCESS. GEORGE OTTE, Rev. de otorrinolaring. **5**:192 (Dec.) 1945.

Otte presents 8 cases of cerebellar abscess in which the history and the progress presented many interesting problems: First, cerebellar abscess is a frequent association of thrombophlebitis, but it may occur in the absence of an involvement of the lateral sinus, as a complication of labyrinthitis, as a cholesteatomatous growth extending through Troutmann's triangle and as a result of osteomyelitis of the temporal bone. Otte believes that the labyrinthine route is the most common route of infection. In consequence, he stresses the importance of watchfulness in cases of chronic suppurative otitis media which may be complicated by either thrombophlebitis or labyrinthitis. Repeated neurologic examinations are indicated, as are studies of the eyegrounds, lumbar punctures, hemograms and observations of any possible changes of the psychic or mental state of the patient. A diagnosis of frank cerebellar abscess following chronic suppurative otitis media is more or less readily established, but should the abscess follow or be associated with any other complication, such as labyrinthitis, thrombophlebitis or meningitis, it is often difficult to make a clearcut diagnosis. The significant diagnostic signs may be divided into four groups: First, those due to intracranial hypertension, such as intensive occipital headaches, bradycardia, papilledema and vomiting; second, those

of cerebral suppuration, such as somnolence, lack of interest, meningitic signs and rapid loss of weight; third, those of cerebellar involvement, such as dysmetria, adiadokokinesis, difficulty in walking and loss of muscular equilibrium; to these must be added, fourth, the evidence of sepsis, such as high temperature and chills, but these are usually associated with the general state of debility, which differs from that seen in thrombophlebitis, in which the patient appears extremely well.

Otte believes that surgical intervention should be done through the mastoidal route for the following reasons: 1. This is the shortest route. 2. It is unnecessary to cause artificial adhesions, by electrocoagulation, between the meninges and the cerebellar surface, since these adhesions have already been formed by the preexisting inflammatory process. 3. The otitic cerebellar abscess is usually nearer the surface of the cerebellum, at the level of the region of the mastoid process, and hence it is more favorable to the establishment of adequate drainage. Should there be associated meningitis, the conjoined use of penicillin and sulfadiazine should be instituted

PERSKY, Philadelphia. [ARCH. OTOLARYNG.]

Diseases of the Gastrointestinal Tract, Liver and Peritoneum

CYSTIC FIBROSIS OF THE PANCREAS. W. DOUGLAS HENDERSON, Arch. Dis. Childhood **20**:179 (Dec.) 1945.

This is a detailed study of cystic fibrosis of the pancreas in a child 22 months old, with observations at necropsy. Points in the diagnosis of cystic fibrosis are: (1) early age of onset; (2) steatorrhea associated with a large appetite, and (3) absence of pancreatic enzymes obtained by duodenal intubation. Treatment with vitamin A and pancreatin has been tried in many cases, but the results have been too indefinite to be of value, and at present treatment of the disease can only be considered as hopeless. It might be worth while to consider treatment with liver extract and vitamin B, as it was noted that there was a drop in the fat content of the stool after seven injections of Heparglandol (a foreign proprietary preparation of liver extract).

KELLY, Milwaukee.

DIAPHRAGMATIC HERNIA IN A SIXTEEN DAY OLD INFANT. LUIS CABRERA PEREZ, FAUSTINO SIRVEN and DAVID GARCIA, Rev. cubana de pediat. **18**:21 (Jan.) 1946.

The authors review the history, symptoms, diagnosis and treatment of diaphragmatic hernia with explanatory notes on the embryologic development of the diaphragm. They present a case with autopsy. It was a left diaphragmatic hernia in a 16 day old girl with dyspnea and cyanosis. There was displacement of the heart to the right. The hernia contained intestines and the left kidney. The diaphragmatic opening did not correspond to any normal hiatus. There was absence of the peritoneal sac. This was a congenital left diaphragmatic hernia of the embryologic type.

KEITH, Rochester, Minn.

CELIAC DISEASE AND CELIAC SYNDROME: CONCEPT AND PATHOGENESIS. M. SUAREZ PERDIGUERO, Rev. espãn. de pediat. **1**:683 (Sept.-Oct.) 1945.

The author bases his study on 17 cases which he has studied. Celiac disease, according to the author's postulation, is seen in babies in the second half year of life and is characterized by the stools but not by a large belly, irritability, dwarfism, osteopathy, etc. Early dietary treatment is important. The celiac syndrome follows infectious disease, parasitism or vitamin or hormonal deficiencies. A gastrointestinal functional insufficiency must be present, however, before these disturbances can precipitate the celiac syndrome.

MILLER, Newark, Ohio.

NEW AQUISITIONS IN THE THERAPY OF INFANTILE INTESTINAL PROCESSES. A. ALLUE HORNA, Rev. espān. de pediat. **1**:719 (Sept.-Oct.) 1945.

This study is based on the effect of administration of Irgafen (N-[3,4-dimethylbenzoyl]-sulfanilamide) to 226 patients with diarrhea. Seventy-eight of these had parenteral infection and 127 dysenteriform enterocolitis; 21 were older children with diarrhea from extradigestive infection. The results obtained seemed to be good, according to the author, better than with sulfaguanadine or sulfathiazole. Unfortunately the organisms causing the diarrhea are not presented and the series is not controlled.

MILLER, Newark, Ohio.

Nervous Diseases

NEURODYNAMICS OF TICS. PAUL H. HOCH, Nervous Child **4**:329, 1944-1945.

The author discusses the possible causes of tics, whether they are on a functional basis or organic and, in the latter case, whether they are cortical or extrapyramidal. He concludes that they may be on either a psychologic or an organic basis; in either case the psychomotor apparatus is impaired. When they are psychologic it indicates that a certain infantilism is present concerning their psychomotor apparatus.

BEVERLY, Oak Park, Ill.

THE SPASTIC'S SPEECH SITUATION. STELLA MCKIBBEN, Quart. J. Speech **31**:358, 1945.

In the lives of persons with cerebral palsy, social environment is a great influence for either good or evil because of its effect on the reactions of nerves and muscles. Specialists believe that they know the importance of social environment, but they often do not take the time to go into a detailed consideration of it in each case. Parents usually believe that the spastic child is shockingly ill and consult many physicians, thus building up abnormal concepts, and the child thus goes through a disastrous environment, which has a direct effect on his condition. Any one working with spastic children should understand the importance of environment and maintain an understanding attitude in helping them to express themselves orally and otherwise.

PALMER, Wichita, Kan. [ARCH. OTOLARYNG.]

AMBLYOPIA WITH HYDROCEPHALUS. K. N. PRADHAN, Indian J. Ophth. **6**:11 (April) 1945.

In a case of acute hydrocephalus, possibly of malarial or filarial origin, in an infant 3 weeks old, there was in the right eye a gray-white atrophy of the optic nerve, and in the left eye congestion of the disk. Two years later there was marked atrophy of the optic nerve in both eyes, though the child could recognize people at 20 feet (6 meters). As modern treatment will save an increasing number of patients with serous meningitis, the problem of preserving the vision of such patients confronts one.

W. ZENTMAYER. [ARCH. OPHTH.]

TREATMENT OF INFLUENZAL MENINGITIS. CONRAD GASSER, Helvet. pædiat. acta **1**:165 (Nov.) 1945.

Treatment of influenzal meningitis is discussed and 7 cases in which there was recovery are reviewed. Decisive for these cures was a high dosage of the sulfonamide compounds at the beginning of the treatment (0.5 Gm. per kilogram of body weight a day). This dose has to be started as soon as the presence of influenzal meningitis is suspected. Oral administration may be allowed in cases of light disease, but in cases of severe disease parenteral administration is necessary, eventually combined with continuous intravenous infusion. In case of doubt purulent leptomeningitis must be treated as influenzal meningitis. If the treatment proves to be unsuccessful or if a relapse occurs, the sulfonamide drug must be

replaced by another one, again given in the same high dose. The higher the amount of sulfonamide drug per day is, the better is the prognosis. In addition daily spinal taps, repeated blood transfusions, administration of vitamins and stimulation are recommended. The treatment has to be surveyed by regular blood controls. Oncoming toxic manifestations must be counteracted by repeated blood transfusions, and furthermore the drug must be changed. Treatment with sulfonamide drugs may not be discontinued until the examination of the spinal fluid shows a normal result and the temperature has become normal.

AUTHOR'S SUMMARY.

Diseases of the Genitourinary Tract

HYDROMETROCOLPOS IN INFANCY: A CAUSE OF URINARY RETENTION, INTESTINAL OBSTRUCTION AND EDEMA OF THE LOWER EXTREMITIES. PAUL MORRIS, Am. J. M. Sc. **210**:751 (Dec.) 1945.

The literature on hydrometrocolpos is reviewed and a case reported with a edema and cyanosis of the lower extremities due to pressure on the pelvic vessels. The author demonstrated that imperforate hymen is not the cause of the vaginal closure but that failure of the perforation of the vagina was due to arrested embryologic development such as one finds in imperforate anus.

The condition is recognized in the newborn infant if the distention of the vagina and the uterus is sufficient to cause pressure on the bladder and urinary retention. Otherwise the condition might not be recognized until just before the menarche when there would again be increased activity of the cervical and uterine glands.

HENSKE, Omaha.

DIAGNOSIS AND TREATMENT OF HYPOTHYROIDISM IN THE INFANT. M. SUAREZ PERDIGUERO, Rev. espãn. de pediat. **1**:649 (Sept.-Oct.) 1945.

The author reviews the clinical picture of hypothyroidism in the infant, stressing the difficulty in diagnosing the disease in its milder form or when the patient is a younger infant. The osseous age is not considered of as great value as in older persons but is used in conjunction with other data. Likewise levels of cholesterol in the blood are frequently not elevated in younger infants with hypothyroidism. Low values for phosphatase and iodine in the blood are considered of great diagnostic value, and the phosphatase in the blood is of value in control of treatment. Early treatment is important, and even in those cases in which the psychologic development is not perfect, the social adaptation of patients treated early is better than in patients treated later. The dosage of thyroid extract should be high at first and then maintained at a lower level, with observation for toxic symptoms.

MILLER, Newark, Ohio.

CHLORINE METABOLISM IN THE NEPHROTIC SYNDROME: ROLE OF CHLORINE IN THE GENESIS OF EDEMA. C. KOUSMINE, Helvet. pædiat. acta **1**:5 (Aug.) 1945.

In many of the children with the nephrotic syndrome whose excretion of chlorides in the urine was studied for months and even for years at the Childrens' Hospital in Zurich, an excessive elimination of chlorides was found after the disappearance of the edema. The author therefore thinks that besides the retention of chlorides in the retained fluid there must be another retention which is independent of the former and may be called the dry one. The chlorides retained in the latter way are probably deposited in the connective tissue. The retention is probably due to an intermediary acidosis, which precedes the formation of edema and appears at the moment when the connective tissue, saturated with chlorides, is incapable of storing more of them. The treatment of this intermediary acidosis by the administration of large doses of sodium bicarbonate has resulted in a considerable amelioration of the condition in an 18 month old patient with the nephrotic syndrome in severe form.

AUTHOR'S SUMMARY.

Diseases of the Ductless Glands; Endocrinology

WATERHOUSE-FRIDERICHSEN SYNDROME: REPORT OF FIVE CASES. FREDRIK WAHLGREN, Nord. med. **15**:2496 (Sept. 5) 1942.

The author reports on 5 cases of the Waterhouse-Friderichsen syndrome. Of the 5 patients 1 was a man and the other 4 were infants aged 6 days, 11 days, 5 months and 7 months. In 2 infants the cause of the adrenal hemorrhage was considered to be an obstetric trauma, while in the other 2 infants the hemorrhage probably was due to meningococcic infection. Two patients did not show cyanosis or petechiae, and 2 patients were found to have hypoglycemic blood (43 and 24 mg. per hundred cubic centimeters, respectively). WALLGREN, Stockholm, Sweden.

Miscellaneous

A COMPARISON OF THE BEHAVIOR OF MICROCRYSTALLINE SULFADIAZINE WITH THAT OF ORDINARY SULFADIAZINE IN MAN. JOHN C. REINHOLD, FRED J. PHILLIPS and HARRISON F. FLIPPIN, Am. J. M. Sc. **210**:141 (Aug.) 1945.

Patients receiving by mouth suspensions of microcrystalline sulfadiazine (3 Gm.) showed significantly higher concentrations of sulfadiazine in serum during the first six hours following its ingestion than did those who received ordinary sulfadiazine.

The excretion of sulfadiazine in the urine of those receiving microcrystalline material was likewise significantly higher during this period. These observations indicate that microcrystalline sulfadiazine is absorbed more rapidly than is ordinary sulfadiazine.

The presence of sucrose or of flavoring material had no influence on the rate of absorption of microcrystalline sulfadiazine or ordinary sulfadiazine.

In fasting subjects, the rate of absorption of microcrystalline sulfadiazine was decreased, but remained greater than that of ordinary sulfadiazine whether in fasting subjects or after meals. No such effect of fasting was observed in subjects given ordinary sulfadiazine.

HENSKE, Omaha.

A SIMPLE TECHNIQUE OF STERNAL MARROW BIOPSY FOR SPREADS AND SECTIONS. ELIZABETH MERTENS, Am. J. M. Sc. **210**:630 (Nov.) 1945.

The author demonstrates by figures and also outlines a simple technic of sternal marrow biopsy which combines the advantages of aspiration and the trephine methods.

HENSKE, Omaha.

News and Comment

GENERAL NEWS

Physicians Certified by the American Board of Pediatrics.—The following physicians were certified as specialists by the American Board of Pediatrics, after examinations held at Buffalo May 10 and 11, 1947.

Dr. Ruth Virginia Berney, Newark, N. J.; Dr. Nils William Bolduan, Santa Barbara, Calif.; Dr. Erika Bruck, Buffalo; Dr. Robert Stewart Cleland, Pasadena, Calif.; Dr. John Henry Dale Jr., New York; Dr. John N. DeHoff, Flushing, N. Y.; Dr. Carl Drayer, Brooklyn; Dr. Anne Mary Drislane, Albany, N. Y.; Dr. Milton Carl Fleish, Hartford, Conn.; Dr. James T. Fowler, Bethesda, Md.; Dr. Lewis M. Fraad, New York; Dr. Kate M. Freund, New York; Dr. Bernard German, Newark, N. J.; Dr. Richard H. Godwin, Rockville Centre, N. Y.; Dr. Bernard Greenberg, Brooklyn; Dr. Clifford Grosselle Grulee Jr., Minneapolis, Minn.; Dr. Francis Woodrow Helfrick, Castaner, Puerto Rico; Dr. Samuel Fredrick Jenkins, New York; Dr. Neil Fremont Lebhar, Westport, Conn.; Dr. Maurice Lenarsky, New York; Dr. William I. Levinstein, Rochester, N. Y.; Dr. Philip Frederick Lustbader, Brooklyn; Dr. Simon B. Mendelsberg, Brooklyn; Dr. Eleanor Kellogg Peck, Poughkeepsie, N. Y.; Dr. Benjamin Reder, Fall River, Mass.; Dr. Edward L. Robinson, Hamilton, Ohio; Dr. George David Rook, New York; Dr. Martha L. Smith, New York; Dr. Mildred Stanley, Tyler, Texas; Dr. Henry B. Strenge, Rochester, N. Y.; Dr. Douglass W. Walker, Laconia, N. H.; Dr. Irving Bernard Wexler, Brooklyn, and Dr. Henry Harcus Work Jr., Buffalo.

The following physicians were certified as specialists by the American Board of Pediatrics, after examinations held at Chicago April 25, 26 and 27, 1947.

Dr. Irving R. Abrams, Chicago; Dr. Cornelius Benton Burns, Orangeburg, S. C.; Dr. Beach M. Chenoweth Jr., Birmingham, Ala.; Dr. Robert Cohen, North Hollywood, Calif.; Dr. James A. Conner, Chicago; Dr. LaMeta Frances Dahl, Denver; Dr. Joseph K. David Jr., Jacksonville, Fla.; Dr. Gerti Dooneief, Brooklyn; Dr. Richard E. Dukes, Urbana, Ill.; Dr. Aaron Robinson Edwards, Ann Arbor, Mich.; Dr. James Nickolas Etteldorf, Memphis, Tenn.; Dr. Donald G. Evans, Seattle; Dr. Elizabeth Gentry, Austin, Texas; Dr. Joseph R. Goran, Denver; Dr. Walter A. Goss Jr., Portland, Ore.; Dr. George Wainwright Griffin, Birmingham, Ala.; Dr. Wesley Robert Heard, San Marino, Calif.; Dr. Helen Dickerson Heinen, Chicago; Dr. Charles W. Ihle Jr., Sioux Falls, S. D.; Dr. Charles Godwin Jennings, Grosse Point, Mich.; Dr. William R. Jones, Columbus, Ga.; Dr. Hilda Knobloch, Ann Arbor, Mich.; Dr. John Harry Lange, Atlanta, Ga.; Dr. John W. Leech, Pittsburgh; Dr. Conway Stone Magee, Lake Charles, La.; Dr. Cornelius Shepherd Meeker, Waukegan, Ill.; Dr. Alberta West Parker, Berkeley, Calif.; Dr. Hugh W. S. Powers Jr., Baltimore; Dr. Donald T. Rendel, Hammond, Ind.; Dr. Mary L. Richardson, Philadelphia; Dr. Julius B. Richmond, Chicago; Dr. Harry Osbourne Ryan, Pasadena, Calif.; Dr. John W. Schonwald, Oakland, Calif.; Dr. B. J. Siebenthal, South Bend, Ind.; Dr. William A. Silverman, New York; Dr. Benjamin Spritz, Brookline, Mass.; Dr. Paul Stone Strong, Los Angeles; Dr. John Dudley Tobin, Minneapolis, Minn.; Dr. David F. Weaver, Davenport, Iowa; Dr. Ross R. Weller, Milwaukee; Dr. Alvin DuBois Wert, Portland, Ore.; Dr. William Kerr Zollinger, Toledo, Ohio, and Dr. Ralph L. Zucker, Toledo, Ohio.

Memorial Hospital for the Treatment of Cancer and Allied Diseases Announces an Extension of Its Children's Diagnostic Services.—An extension of the diagnostic service of the Children's Tumor Registry of the Memorial Hospital for the Treatment of Cancer and Allied Diseases was made on Jan. 1,

1947, with the establishment of the Children's Diagnostic Clinic. This service is offered to the pediatric patients of hospitals and private physicians for the diagnosis of neoplastic diseases and is available for ambulant or bed patients. The investigations will be performed at cost or without charge if the patient is unable to afford this. On completion of the study the patient will be referred back to the physician or institution interested, with a report of the findings. If therapeutic recommendations are indicated and requested they will also be made.

Appointment for such examinations may be made by mail or telephone with The Secretary, Pediatric Service, Memorial Hospital, 444 East 68th Street, New York 21, N. Y.

Book Reviews

Currents in Biochemical Research. Edited by David E. Green, M.D. Thirty-One Essays Charting the Present Course of Biochemical Research and Considering the Intimate Relationship of Biochemistry to Medicine, Agriculture and Social Problems. Price, $5. Pp. 486, with illustrations. New York: Interscience Publishers, Inc., 1946.

The editor of this recently published book, David E. Green, expresses in the preface his hope that this survey by some thirty research workers may assist biochemists, chemists and medical doctors in seeing biochemistry in clearer perspective and in its proper relation to other fields of inquiry. The reviewer is here concerned with the book as it may assist medical doctors, especially pediatricians, who occupy a position either as outright biochemists or a position somewhere between physicians and surgeons on the one hand and biochemists on the other.

The contributors have been chosen well, though a few have not measured up to expectations. Essays in this category are those entitled "Immunochemistry," "Chemotherapy," "Viruses," "Biochemical Aspects of Pharmacology" and the "Biological Significance of Vitamins." In some there is popularization in style, if not in content, which belies the statement of the editor that no popularizations were intended; in others the subject is familiar, even stale, but is certainly not current in biochemical research. The eight essays that would seem to be specially stimulating to practicing pediatricians are those on "The Gene and Biochemistry," "Hormones," "Mucolytic Enzymes," "The Steroid Hormones," "Chemical Mechanism of Nervous Action," "Some Aspects of Biochemical Antagonism" and "Some Biochemical Problems Posed by a Disease of Muscle," this last essay having been contributed by the late Dr. Charles Hoagland. The other essays are of prime interest mainly to research biochemists, but there are three in this group which are so beautifully presented as to excite the interest of every one in the science and art of medicine. These three most noteworthy essays are "Fundamentals of Oxidation and Reduction," by Leonor Michaelis, "Viscometry in Biochemical Investigations," by Max A. Lauffer, and "Mesomeric Concepts in the Biological Sciences," by Herman M. Kalckar. Dr. Kalckar's essay may, as he puts it, contain a questionable proportion of speculation; on the other hand, it is a simple, concise and provocative statement of the correlation of catalysis, energy and the theory of resonance, as they apply to biologic problems.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof. S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary-General: Dr. L. Emmett Holt Jr., 477 First Ave., New York 16.
Canadian Committee:
Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto 2.
Secretary: Dr. S. G. Ross, 1390 Sherbrooke St. W., Montreal.
Place: New York. Time: July 14-17, 1947.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

Pan American Congress of Pediatrics

President: Dr. Felix Hurtado, Habana, Cuba.
Place: Mayflower Hotel, Washington, D. C. Time: July 9-12, 1947.

Save the Children International Union

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 43 Quai Wilson, Geneva, Switzerland.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

Associação Paulista de Medicina, Secção de Pediatrias

President: Dr. Vicente Lara.
First Secretary: Dr. Armando de Arruda Sampaio.
Second Secretary: Dr. Luiz Augusto Monteiro Toledo, Av. Brigadeiro Luiz Antonio 393, 1° Andar, São Paulo, Brazil.
Time: Twelfth of every month, 8:30 p. m.

British Paediatric Association

President: Prof. G. B. Fleming, 13 Lynedoch Crescent, Glasgow.
Joint Secretary: Prof. Alan Moncrieff, Hospital for Sick Children, Great Ormond St., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. Gjørup, Dronning Louises Børnehospital, Copenhagen.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

NEDERLANDISCHE VEREENIGING VOOR KINDERGENEESKUNDE

President: Dr. J. L. Keyzer, 114 St. Josephstr., Tilburg.
Secretary: Dr. H. Reerink, 2B Paul Krugerstr., Arnheim.
Place: Children's Hospital or Pediatric Clinic. Time: Three times a year.

PALESTINE JEWISH MEDICAL ASSOCIATION, SECTION OF PHYSICIANS OF CHILDREN'S DISEASES

President: Prof. L. F. Meyer, 29 Toelsohn St., Tel Aviv.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

ROYAL SOCIETY OF MEDICINE, SECTION FOR THE STUDY OF DISEASES IN CHILDREN

President: Prof. Norman Capon, 49A Rodney St., Liverpool, England.
Secretary: Dr. J. N. O'Reilly, 46 Harley St., London W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

SOCIEDAD BOLIVIANA DE PEDIATRIA

President: Dr. Ernesto Trigo Pizarro.
Secretary: Dr. Carlos Ferrufino, Casilla 272, La Paz, Bolivia.
Place: La Paz, Bolivia.

SOCIEDAD CUBANA DE PEDIATRIA

President: Dr. Teodosio Valledor, Habana.
Secretary: Dr. Carlos Hernandez Miyares, Hospital Nuestra Señora de las Mercedes L y 21, Apartado 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Nuestra Señora de las Mercedes y Hospital Municipal de Infancia, Habana. Time: Last Wednesday of every month.

SOCIEDAD DOMINICANA DE PEDIATRIA

President: Dr. Alberto Peguero, Ciudad Trujillo, R.D.
Secretary-Treasurer: Dr. Jaime Jorge, Ciudad Trujillo, R.D.
Place: Children's Ramfis Hospital. Time: 8 p. m., last Thursday of every month.

SOCIEDAD ECUADORIANA DE PEDIATRIA

President: Dr. Francisco de Icaza Bustamente.
Secretary: Dr. M. I. Gómez Lince, P. O. Box 693, Guayaquil.

SOCIEDAD MEXICANA DE PEDIATRIA

President: Dr. Luis Berlanga Berumen, Bucareli No. 85, Altos, Mexico.
Secretary: Dr. Jésus Alvarez de los Cobos, Ignacio Mariscal 124, Mexico.

SOCIEDAD DE PEDIATRÍA DE CÓRDOBA

President: Dr. Juan Francisco Herrera, Córdoba, Argentina.
Secretary: Dr. Bernardo Serebrinsky, Dean Funes 2000, Esq. Ocaña (N.), Córdoba, Argentina.
Place: Comite pro Defenso del Niño, Dean Funes 2000, Córdoba, Argentina. Time: Once a month.

SOCIEDAD DE PEDIATRIA DEL LITORAL

President: Dr. Jose E. Celoria, Hospital Centenario, Rosario, Argentina.
Secretary: Dr. Gloria Lovell, Hospital Centenario, Rosario, Argentina.

SOCIEDAD DE PEDIATRÍA DE SANTIAGO DE CUBA

President: Dr. Antonio Béguez César, Sagarra 107, Santiago de Cuba.
Secretary: Dra. Susana Texdó Vaillant, Padre Pico 57, Santiago de Cuba.
Place: Colegio Médico. Time: Last Tuesday of every month.

Sociedad Venezolana de Puericultura y Pediatria

President: Dr. Pastor Oropeza, Caracas.
Secretary: Dr. Carlos E. Castillo, Hospital Municipal de Niños, Caracas.
Place: Hospital Municipal de Niños Jose Manuel de los Rios, Caracas.

Société de Pédiatrie de Paris

President: Dr. Leveuf, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue Pré aux Clercs, Paris, France.
Place: Hôpital des Enfants Maladies, 149 Rue de Sèvres. Time: 4:30 p. m., third Thursday of every month.

Swedish Medical Society, Section for Pediatrics and School Hygiene

President: Prof. Arvid Wallgren, 53 Strandvägen, Stockholm.
Secretary: Dr. Henning Magnusson, 58 Odengatan, Stockholm.
Place: Various clinics and hospitals in Stockholm and Uppsala. Time: Second Friday of each month (except summer).

Uruguayan Society of Pediatrics

President: Dr. Jose Bonaba, Montevideo.
Secretary: Dr. Héctor Fossatti, Durazno 1882, Montevideo.
Place: Club Medico del Uruguay, Avenida Agraciado 1464 (Piso 13), Montevideo.

NATIONAL

American Medical Association, Scientific Assembly, Section on Pediatrics

Chairman: Dr. John Aikman, 184 Alexander St., Rochester, N. Y.
Secretary: Dr. Gilbert J. Levy, 188 S. Bellevue Blvd., Memphis, Tenn.

American Academy of Pediatrics

President: Dr. Jay I. Durand, 515 Cobb Bldg., Seattle, Wash.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.

American Hospital Association, Massachusetts Hospital Association

Chairman: Dr. Frank Wing, Boston.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly 79, Mass.

American Pediatric Society

President: Dr. Harold K. Faber, 2361 Clay St., San Francisco.
Secretary-Treasurer: Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12.

Canadian Society for the Study of Diseases of Children

President: Dr. H. S. Little, 300 Dufferin Ave., London, Ontario.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, 67 College St., Toronto 2.

Society for Pediatric Research

President: Dr. Donovan J. McCune, 3975 Broadway, New York.
Secretary: Dr. Robert Ward, Bellevue Hospital, New York 16.

SECTIONAL

Intermountain Pediatric Society

President: Dr. Wesley H. Anderson, 2404 Washington Blvd., Ogden, Utah.
Secretary-Treasurer: Dr. F. M. Kriete, State Capitol, Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

New England Pediatric Society

President: Dr. James Marvin Baty, 1101 Beacon St., Brookline, Mass.
Secretary-Treasurer: Dr. Charles D. May, 300 Longwood Ave., Boston.
Place: Boston Medical Library. Time: Three meetings a year, occurring from September to May.

North Pacific Pediatric Society

President: Dr. Percy Guy, 502–2d Ave., Seattle, Wash.
Secretary: Dr. Aldis B. Johnson, 515 Cobb Bldg., Seattle, Wash.

Northwestern Pediatric Society

President: Dr. George Kimmel, Redwing, Minn.
Secretary-Treasurer: Dr. Northrop Beach, 1009 Nicollet Ave., Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Rocky Mountain Pediatric Society

President: Dr. Jackson L. Sadler, 109 W. Olive St., Ft. Collins, Colo.
Secretary: Dr. Edwin T. Williams, 1850 Gilpin St., Denver 6.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. Stewart H. Welch, 117 S. 20th St., Birmingham 5, Ala.
Secretary: Dr. W. Ambrose McGee, 1601 Monument Ave., Richmond 20, Va.

STATE

Alabama Pediatric Society

President: Dr. Jerome C. Chapman, 2160 Highland Ave., Birmingham.
Secretary-Treasurer: Dr. Ruth Berrey, 2021–6th Ave. N., Birmingham.

Arizona Pediatric Society

President: Dr. W. P. Sherrill, 342 W. McDowell Rd., Phoenix.
Secretary: Dr. W. F. Schoffman, 926 E. McDowell Rd., Phoenix.
Place: Phoenix or Tucson. Time: At time of state meeting.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. Charles W. Leach, 2000 Van Ness Ave., San Francisco 9.
Secretary: Dr. Chester I. Mead, 1930 Truxton Ave., Bakersfield.

Florida State Pediatric Society

President: Dr. James R. Boulware, Lakeland.
Secretary: Dr. Hugh A. Carithers, 2000 Park St., Jacksonville 4.
Place: Concurrent with state association convention and fall meeting.

Georgia Pediatric Society

President: Dr. Alton Johnson, 102 E. Central Ave., Valdosta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
Place: Atlanta. Time: At time of annual state meeting.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. David Gaberman, 1179 Allyn St., Hartford, Conn.
Secretary: Dr. Herman Yannet, Southbury Training School, Southbury.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. Gerard N. Krost, 2376 E. 71st St., Chicago.
Secretary: Dr. Frederick H. Maurer, 331 Fulton St., Peoria 2.

Indiana State Pediatric Society

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Matthew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Arnold M. Smythe, 406–6th Ave., Des Moines.
Secretary-Treasurer: Dr. James Dunn, 414 Blackhawk Hotel, Davenport.

Louisiana State Pediatric Society

President: Dr. S. George Wolfe, 1560 Line Ave., Shreveport.
Secretary-Treasurer: Dr. W. C. Rivenbark, 1448 Louisiana Ave., New Orleans 15.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. Carl H. Laws, 12 Pierrepont St., Brooklyn, N. Y.
Secretary: Dr. George R. Murphy, 292 Madison Ave., New York.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. Joseph A. Gilmartin, 3700–5th Ave., Pittsburgh 13.
Secretary: Dr. Carl C. Fischer, 100 W. Coulter St., Philadelphia 44.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Donald J. Barnes, 3001 W. Grand Blvd., Detroit, Mich.
Secretary: Dr. Mark F. Osterlin, Central Michigan Children's Clinic, Traverse City.

Mississippi State Pediatric Society

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

Nebraska Pediatric Society

President: Dr. Paul N. Morrow, 1614 Medical Arts Bldg., Omaha 2.
Secretary-Treasurer: Dr. Michael Crofoot, 1407 Medical Arts Bldg., Omaha 2.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

New Mexico State Pediatric Society

President: Dr. M. K. Wylder, 221 W. Central Ave., Albuquerque.
Secretary-Treasurer: Dr. Ly Werner, 221 W. Central Ave., Albuquerque.

North Carolina Pediatric Society

President: Dr. Charles R. Bugg, Professional Bldg., Raleigh.
Secretary: Dr. Charles F. Williams, 817 Hillsboro St., Raleigh.

Northern California Pediatric Society

President: Dr. Edith M. Meyers, 431–30th St., Oakland.
Secretary: Dr. A. L. Gleason, 411–29th St., Oakland 9.
Place: San Francisco. Time: Second Thursday of February, April, September and November.

Oklahoma State Pediatric Society

President: Dr. J. B. Snow, 625 N. W. 10th St., Oklahoma City.
Secretary: Dr. Bertha Levy, 1200 N. Walker St., Oklahoma City.
Place: With Oklahoma State Medical Association. Time: Annual Meeting.

South Carolina Pediatric Society

President: Dr. George D. Johnson, Spartanburg.
Secretary-Treasurer: Dr. B. O. Ravenel. Roper Hospital, Charleston.

Tennessee Pediatric Society

President: Dr. F. T. Mitchell, 376 S. Bellview, Memphis.
Secretary-Treasurer: Dr. W. O. Vaughan, 2103 Hayes St., Nashville 5.
Time: Second Monday in April.

Texas Pediatric Society

President: Dr. Max Woodward, 202 N. Travis St., Sherman.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas 4.

Virginia Pediatric Society

President: Dr. Emily Gardner, 1100 W. Franklin St., Richmond 20.
Secretary: Dr. Louise Galvin, 214 S. Boulevard, Richmond 21.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. Jack Basman, 1021 Quarrier St., Charleston.
Secretary: Dr. Harry E. Baldock, Medical Arts Bldg., Charleston.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. J. I. Hartman, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. Alexander N. Freed, 2012 W. 25th St., Cleveland.
Place: Cleveland Medical Library Bldg. Time: 8:15 p. m., October, December, February and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. J. H. Ebbs, 13 Queen's Park, Toronto, Canada.
Secretary: Dr. J. F. McCreary, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine. Time: 8:30 p. m., third Thursday of each month.

Baltimore City Medical Society, Pediatric Section

Chairman: Dr. Charles O'Donovan Jr., 5 E. Read St., Baltimore.
Secretary: Dr. T. Campbell Goodwin, 16 E. Biddle St., Baltimore.

Bronx Pediatric Society

President: Dr. Harry S. Pizer, 1726 Davidson Ave., New York.
Secretary: Dr. Nathan M. Greenstein, 1488 Metropolitan Ave., New York.
Place: Morrisania City Hospital. Time: 8:30 p. m., second Wednesday of each month, except June, July, August and September.

Brooklyn Academy of Pediatrics

President: Dr. George E. Brockway Jr., 176 Fenimore St., Brooklyn.
Secretary: Dr. Bernard Benjamin, 845 Park Pl., Brooklyn 16.
Place: Granada Hotel. Time: 8:45 p. m., fourth Wednesday of February, March, April, October and November.

Buffalo Pediatric Society

President: Dr. Douglas P. Arnold, 159 Bidwell Parkway, Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest Ave., Buffalo 9, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

Central New York Pediatric Club

President: Dr. Arthur W. Benson, 2 St. Paul St., Troy.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Places: Various cities in New York. Time: Third Tuesday of April and October.

Chicago Pediatric Society

President: Dr. H. William Elghammer, 2371 E. 71st St., Chicago.
Secretary: Dr. Alvah L. Newcomb, 723 Elm St., Winnetka, Ill.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May inclusive.

Cincinnati Pediatric Society

President: Dr. Leo S. Friedman, 666 Rockdale Ave., Cincinnati 29.
Secretary: Dr. George Renner, 3144 Jefferson Ave., Cincinnati.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Dallas Pediatric Society

President: Dr. Shirley Hodges, 2703 Oak Lawn Ave., Dallas, Texas.
Secretary-Treasurer: Frances M. Love, 4585 Belfort, Dallas 4.
Place: Children's Medical Center. Time: 1 p. m., third Saturday and 8:30 p. m., the first Thursday of each month.

Detroit Pediatric Society

President: Dr. Otto Grob, 651 Fisher Bldg., Detroit 2, Mich.
Secretary: Dr. Harold B. Rothbart, 773 Fisher Bldg., Detroit 2, Mich.
Place: Variable. Time: Third Wednesday of each month.

Houston Pediatric Society

President: Dr. George Salmon, 1220 Bartlett St., Houston, Texas.
Secretary: Dr. Betty Moody, 526 Richmond Rd., Houston, Texas.
Place: River Oaks Country Club. Time: 7:30 p. m., last Tuesday of each month

Kansas City (Missouri) Pediatric Society

President: Dr. Harry M. Gilkey, 1624 Professional Bldg., Kansas City.
Secretary: Dr. Leland Speer, 1022 Parkway, Kansas City.
Place: Kansas City General Hospital. Time: On call.

Knoxville-Oak Ridge Pediatric Society

President: Dr. Henry S. Christian, 205 Medical Arts Bldg., Knoxville, Tenn.
Secretary: Dr. John Eggleston, Oak Ridge Hospital, Oak Ridge, Tenn.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles 26.
Secretary-Treasurer: Dr. Robert Simonds, 267 S. Beverly Dr., Beverly Hills, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Henry Rascoff, 895 Eastern Parkway, Brooklyn.
Secretary: Dr. Harold Levy, 750 St. Marks Ave., Brooklyn.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., fourth Monday of each month.

MEDICAL SOCIETY OF THE COUNTY OF QUEENS, INC., SECTION ON PEDIATRICS

Chairman: Dr. Meyeron Coe, 217-02-91st Ave., Queens Village, N. Y.
Secretary-Treasurer: Dr. Louis Appel, 143-42 Cherry Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON PEDIATRICS

President: Dr. John Washington, 2203 Wyoming Ave., Washington, D. C.
Secretary-Treasurer: Dr. George Maksim, 1418 Good Hope Rd. S.E., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., third Thursday of October, January, March and May.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. Kinsey M. Buck, 1193 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Barton Etter, 1024 Madison Ave., Memphis, Tenn.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. G. H. Fellman, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. R. P. Showalter, 411 E. Mason St., Milwaukee 2.
Place: Milwaukee Children's Hospital. Time: Second Wednesday in February, April, June, October and December.

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Chairman: Dr. Robert O. Du Bois, 122 E. 76th St., New York.
Secretary: Dr. Harold W. Dargeon, 1095 Park Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from November to May, inclusive, 8:30 p. m.

OKLAHOMA CITY PEDIATRIC SOCIETY

President: Dr. J. B. Snow, 625½ N. W. 10th St., Oklahoma City.
Secretary: Dr. Bertha Levy, 1200 N. Walker St., Oklahoma City.
Place: Oklahoma Club. Time: Third Thursday of each month.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Arthur M. Dannenburg, 235 S. 15th St., Philadelphia 2.
Secretary: Dr. D. Stewart Polk, Rosemont, Pa.
Place: College of Physicians, 19 S. 22d St. Time: 8:30 p. m., second Tuesday from October to May, inclusive.

PITTSBURGH PEDIATRIC SOCIETY

President: Dr. Elwood Stitzel, 403 Central Trust Bldg., Altoona, Pa.
Secretary-Treasurer: Dr. C. J. Stoecklein, Medical Arts Bldg., Pittsburgh 13.
Place: Pittsburgh Academy of Medicine. Time: Second Friday in October, December, February and April.

RICHMOND PEDIATRIC SOCIETY

President: Dr. Edwin L. Kendig Jr., 828 W. Franklin St., Richmond 20, Va.
Secretary-Treasurer: Dr. Thomas S. Chalkley, 1200 E. Broad St., Richmond, Va.
Place: Medical College Hospital. Time: 8 p. m., third Thursday of each month, except June, July and August.

Rochester Pediatric Society

President: Dr. Jerome Glaser, 300 S. Goodman St., Rochester 7, N. Y.
Secretary-Treasurer: Dr. Daniel B. Peeler, 1162 Lake Ave., Rochester 13, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee.
Time: Third Friday of each month from October to May.

St. Louis Pediatric Society

President: Dr. Joseph C. Jaudon, 337 N. Euclid Ave., St. Louis.
Secretary-Treasurer: Dr. Gilbert B. Forbes, 500 S. Kingshighway, St. Louis 10.
Time: Second Thursday of each month from October to May, inclusive.

San Antonio Pediatric Society

President: Dr. Sidney R. Kaliski, 518 Moore Bldg., San Antonio 5.
Secretary-Treasurer: Dr. Edith M. Bonnet, 228 Medical Arts Bldg., San Antonio 5.
Place: Variable. Time: 7:30 p. m., first Tuesday of every month.

Seattle Pediatric Society

President: Dr. Norman W. Murphy, 1300 Madison St., Seattle 4.
Secretary: Dr. David M. Harris, 630 Medical Arts Bldg., Seattle 1.
Place: College Club. Time: Third Friday of each month from September to June at 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Donald C. Shelby, 416 N. Bedford Dr., Beverly Hills, Calif.
Secretary: Dr. Harry F. Dietrich, 416 N. Bedford Dr., Beverly Hills, Calif.
Place: University Club of Los Angeles. Time: 6:30 p. m., first Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Park Bradshaw, Muskegon, Mich.
Secretary: Dr. Harry A. Towsley, 1313 E. Ann St., Ann Arbor, Mich.

Westchester County Medical Society, Pediatrics Section (New York)

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